THE
LARYNGOSCOPE.

VOL. XLVII SEPTEMBER, 1937. No. 9

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

B.—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

THE DIAGNOSIS OF OCCUPATIONAL OR TRAUMATIC DEAFNESS; A HISTORICAL AND AUDIOMETRIC STUDY.*†

C. C. BUNCH, Ph.D., St. Louis.

INTRODUCTION.

The descriptions of the results of the functional tests which appear in the textbooks are insufficient to permit the differential diagnosis of occupational deafness. The records of such cases are usually tabulated and filed with those having other etiology. The patients themselves may often receive treatment which should have been prescribed for other forms of deafness. One is not justified in attributing deafness to toxic foci, to acute infection, or to advancing age because the functional tests reveal a loss of hearing for high tones. Neither is he justified in attributing the loss of hearing in an aviator to occupational influences when his functional tests reveal the Bezold triad for otosclerosis.

This study was inaugurated in the hope that it might bring some light on this important clinical and industrial subject.

*Read as part of the Symposium, "The Neural Mechanism of Hearing," at the Seventieth Annual Meeting of the American Otological Society, Long Beach, N. Y., May 28, 1937.

†From the Oscar Johnson Institute, Department of Otolaryngology, Washington University Medical School, St. Louis.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 17, 1937.

Industrial America today is rapidly growing more and more noisy. The last 30 years have seen an enormous increase in noise-producing machinery. With these great changes in the industrial world, is the human ear adapting itself to compensate for the changes in environment? Evolutionists may grant that organic changes may take place, but only with the passing of many generations. It seems more probable that the hearing organ is "taking it on the chin." One has but to note the absence of studies relating to this subject in today's journals to convince himself that it is often unrecognized or ignored.

The proof of the actual relationship between the acoustic trauma and deafness must ultimately be derived from specific studies. This implies that the hearing of employees shall be tested before they are employed and at intervals during their employment, care being taken to eliminate factors other than those truly occupational which may have an adverse influence on hearing. It seems improbable at the present time that such a program will be instituted even on a small scale because of the labor and expense involved. As a substitute for this scientific approach, certain investigators have tested the hearing of employees who have worked in noisy surroundings and compared the results with those secured from tests of those working under conditions of quiet. In such investigations, the number of employees tested should be sufficiently great that the results shall be an adequate "sampling" of the whole of both groups before definite conclusions may be drawn. A third method is that of investigating individual cases. This method is probably less accurate, from the scientific standpoint, than either of the first two mentioned, for it assumes that histories are accurate, an assumption which all clinicians know has its limitations, and depends upon the skill of the examiner in securing the history. Regardless of these very evident sources of error, it must be said that much of our information in all fields of medicine has been secured in this manner and the method should not be regarded lightly. A fourth method open for investigation is that of animal experimentation. This method is also open to grave error; first, because no absolutely accurate method for determining the auditory acuity of animals has been devised; second, it is quite difficult to maintain animals in health under controlled conditions for a time com-

parable with that under which men are working; and third, it is not known that the ears of animals and men react in the same manner under experimental conditions.

Industrial deafness, as such, has within recent years received rather scant attention by the otological world. Laymen, on the other hand, are aware of some of the ill effects of noise. Office managers know that their employees make fewer mistakes, become less fatigued and do better and faster work under conditions of quiet. Certain cities have passed ordinances regulating the hours during which automobile horns, compressed-air hammers and loud speakers may be used. Factory employees work with their ears stopped with plugs of cotton. Zones of quiet are established about hospitals. It is probably true that some of these anti-noise campaigns are inaugurated or abetted by companies who expect to profit from the sale of sound-proofing materials, but it is evident that there is an active campaign against the terrible din of our city streets. It can be said with certainty that no one knows to what extent these street noises adversely effect hearing, for no studies of this nature have been made. Gilbert,[59] a medical inspector of factories in Belgium, reviewed this subject in 1921 and stated, "The influence of noise, which is at times deafening, has been little studied in spite of the fact that it is present in nearly all industries. . . . It seems wise . . . to reintroduce so important a subject in order to stimulate research on the part of ear specialists. . . . Classic writers on occupational diseases are generally reticent on the subject of disorders caused by noise in industry." He suggests, "An international commission composed of doctors having access to all the factories in the country ought to decide the basis of all inquiry into industrial sounds, for the purpose of preparing a detailed account of the principal noisy industries."

Workmen who accept employment, accept also the hazards which are incumbent upon that occupation. These are usually recognized and provision is made for the protection of the workmen. New hazards are discovered from time to time and additional protective devices installed. Industrial hazards to hearing, however, have been recognized for years, but in most cases, like Mark Twain and the weather, everybody talks about it, but nobody does anything. Just why the hear-

ing of workmen has not universally received protection is probably due to the difficulty in establishing proof of the cause and effect relationship. This deafness is usually unrecognized in its earliest forms by either the workman, his employer or the otologist; its progress is often not unlike that of other forms of deafness; its classically described characteristics are so like the deafness from other causes that its recognition may be difficult.

There are probably two reasons why extensive programs of repeated tests in industries have not been established. First, the employer is inclined to discourage such an investigation because of the possibility of legal regulations. Recently an employer, when asked what his reaction would be to such a program in his factory, replied, "My first reaction would be to let sleeping dogs lie." Second, the employee himself may oppose it for fear some handicap will be made known and his tenure be made insecure. We are informed that there is one exception to this. Certain railroads require frequent examinations of the hearing of employees who are responsible for the safety of the public. If there be such tests in America, no audiometric studies have been reported. Many industries accept employees with defective hearing, the only requisite being that the applicant shall hear well enough to receive the spoken or shouted orders of the foreman and be able to perform his duties. Workmen are recruited from the ranks of those who are leaving our schools and tests have shown that from 4 to 10 per cent of these are hard-of-hearing. It is little wonder that courts have no factual basis upon which they may justly award compensation for damages to hearing which result from industrial hazards.

OCCUPATIONAL DEAFNESS—EVIDENCE FROM THE TEXTS.

The first reference to this subject which the writer could find in the English language was by Fosbroke,[54] who wrote in 1831, as follows: "Other causes of deafness are local and mechanical, as in deafness caused by continuous noise, as blacksmiths' deafness. . . . The blacksmiths' deafness is a consequence of their employment; it creeps on them gradually, in general at about 40 or 50 years of age. At first the patient is insensible of weak impressions of sound; the deafness increases with a ringing and noises in the ears. . . . *It*

has been imputed to a paralytic state of the nerve occasioned by the noise of forging by certain modern writers, and by old writers, to permanent overtension of the drum membrane." (Italics by writer.) He did not name either the "old" or the "modern" writers, nor did he refer to any special investigation. It is evident that the pathology produced in the hearing organ was at that time a matter of dispute. A survey of our *modern* writers shows that there is, in 1937, no entire agreement.

Considerable evidence as to the cause and effect relationship must have been apparent to later writers, for frequent reference is found in more recent textbooks. Toynbee,[144] in 1860, stated: "Deafness from concussion is of three modes: *1.* Blows on the ear. *2.* Loud sounds. *3.* Falls." Dalby[42] mentioned the subject in his lectures of 1872, in this manner: "Deafness may, however, follow repeated shocks as instanced in the case of boilermakers." Roosa,[126] in 1873, summarized his observations as follows: "Workmen employed in hammering large iron plates such as are used in making boilers of steam engines, are very apt to lose much of their hearing power. I am informed by the superintendents and workmen of some such factories, that a large proportion of the men who have been long in the horrid din of a boiler shop, become deaf. So many of these cases were found, that at one time boilermakers' deafness figured as a separate disease of the ear in the statistical reports of one of our institutions where aural diseases were treated. Examination of such cases has shown me that the lesion causing the impairment of hearing and deafness must be sought for in the labyrinth, and it is probably due to concussion of the fibres of the nerve in the cochlea and the semicircular canals." Hartmann,[72] in 1887, described this deafness more definitely. He stated, "Bone-conduction is considerably diminished, the two highest tones not being heard at all (c^4 and g^4). This shows that the injurious action of the noises in boiler shops is chiefly expended upon those portions of the sound perceiving apparatus, which serve for the perception of the higher tones. Field,[53] in 1893, stated: "*Artillerymen and boilermakers,* being frequently exposed to concussive shocks, are liable to chronic inflammation of the internal ear, coming on gradually." Bruhl,[23] in 1892, stated: "At the upper limit of the scale, deafness for high sounds, such as the sound of 's' or the chirping of crickets, is met with in nervous deafness, especially in *old*

persons and in boilermakers, owing to the atrophy of the nerve fibres in the base of the cochlea." (Italics by writer.) Hartmann and Bruhl used tuning forks of definite pitch in differential diagnosis and understood, in part at least, the tonal characteristics of the voice and of sounds from other sources. It is evident, too, that the similarity between the deafness caused by excessive sounds and that encountered in people of advanced age was recognized. The more recent texts, as a rule, appear almost equally reticent in describing the clinical picture of occupational deafness. The descriptions would lead one to think that almost any deaf patient who gave a history of having been employed in a noisy place would automatically be diagnosed as having boilermakers' deafness, no matter what the tuning fork tests or the otoscopic examination revealed. On the other hand, should the patient's history reveal no evidence of the trauma of occupational noises, it seems probable that he might receive treatment for other types of deafness when the real etiological factor rested in an unknown occupational environment or other acoustic insult. Bishop[16] gives little information. In his chapter on "Diseases of the Internal Ear," he states: "Many workers in boiler shops have appeared at the clinics with great dullness of hearing and tinnitus. Their ears were generally full of hardened, impacted plugs of black wax. After removing these the impairment of hearing still remained of a high degree. Blacksmiths, tinsmiths, coopers and ironworkers suffer similarly. This is due to the constant concussion of the drumhead, ossicles and the intralabyrinthine fluid and the auditory nerve from their incessant hammerings. The effect is to produce, in addition to the labyrinthal affection, the sclerotic form of middle-ear catarrh." Ballenger,[4] in a section on "Injuries to the Labyrinth; Concussion of the Labyrinth," mentions sudden condensation of the air by an explosion or a blow of the hand causing injury to the ear, either with or without rupture of the drum membrane. He states that the "hearing for high tones is lost." In the section on "Occupational Deafness," he also states: "For many years it has been recognized that among those who have been engaged in certain occupations for a long time, especially where continuous or frequently recurring sounds are heard, there is apt to be more or less deafness. The terminal nerve filaments of the labyrinth are continously subjected to irri-

tation and undergo a degenerative change often amounting to complete atrophy, and subsequent deafness." Is one to understand from these statements that the hearing is uniformly diminished for all tones? Turner,[148] in a section entitled, "Traumatic Affections of the Auditory Nerve and Labyrinth," has a paragraph under the heading of "Noise Deafness" which states that it "is met with in boilermakers, coopers, factory workers, artillerymen and sailors in the Royal Navy." The description of this deafness, according to Turner, is as follows: "The deafness increases gradually, and is not as a rule accompanied by tinnitus. . . . Sportsmen occasionally suffer from deafness in one ear—as a rule the left." Bacon,[2] in his section on "Boilermakers' Deafness," states: "From all the cases that have come under my observation, tests made with the tuning forks have demonstrated the fact that there was impaired bone-conduction, showing that the disturbance of hearing and tinnitus were not due primarily to middle-ear inflammation." Lake and Peters[94] are more specific. They state: "Their deafness has a well marked peculiarity; *viz.*, they can hear comparatively well in the clash and crash of the shops. Voice and whisper are very much diminished; Rinné with the c(128 d.v.) is positive; bone-conduction is reduced to one-third or less of normal." Phillips[112] mentions boilermakers' deafness only when discussing paracusis. Dench,[44] McAuliffe,[105] and Jackson and Coates[81] do not mention this subject. Syme[143] says: "Nerve deafness is found markedly in those who work continuously in loud noises—such as boilermakers." Kerrison[87] recognized boilermakers' deafness as being "of a type characteristic of a labyrinthine or nerve lesion." Bezold and Siebenmann[12] state: "*The most frequent injury to the inner ear is that from excessive noise, the acoustic trauma,*" and differentiate these trauma cases into two groups: "*1.* A form of injury to the labyrinth caused by one short sound like a detonation, explosion, whistle of a locomotive. *2.* A form caused by frequently repeated loud noises." Politzer[115] is much more descriptive and definite. He says, "In affections of the ear induced by the various trades, the results of the tuning forks are characteristic of a primary diseased condition of the nerve; the perception through the cranial bones is greatly diminished, and Rinné's test positive; furthermore, there is defect in the perception for the upper range of the scale, as well as for the tones of Galton's

whistle." His investigations "led him to conclude that, next to locksmiths, boilermakers and coopers are most frequently afflicted with a disturbance in hearing. . . . Concussions of the terminal filaments of the acoustic nerve are brought about by the sudden condensation of the air in the external meatus, or by the action of intense noises in the immediate neighborhood of the ear; the former are most frequently caused by a blow on the ear, while the latter are produced by violent detonations. In cases in which the condensation of the air in the meatus is due to a box on the ear, the concussion of the labyrinth is much more intense in those instances in which the membrana tympani remains intact, because the entire force of the blow is transmitted by the footplate of the stapes to the labyrinth; in those cases, however, in which the membrane is ruptured, the greater portion of the active force is expended in causing the rupture."

BOILERMAKERS' DEAFNESS—EVIDENCE FROM ORIGINAL STUDIES.

Holt,[76] in 1882, using a watch, tested the hearing of 40 men who had been employed in making steam boilers. Their ages ranged from 19 to 62 years. The normal distance for this watch was two meters. Seven could not hear the watch at all, 23 heard it when it was pressed against, close to or at a few centimeters from one ear or the other. Ten who had worked at this occupation but a short time could hear it at from 10 to 100 cm. He stated, "All thus engaged become more or less deaf, the degree and length of time elapsing before this occurs depending somewhat upon the tendency of the middle ear to take on catarrhal inflammation." Barr,[5] in 1886, reported one of the most complete studies of this kind. Because of the inaccessibility of his report, it is reviewed here in some detail. He attempted to determine four things: *1.* the extent of the hearing loss; *2.* the region of the ear affected; *3.* the course of the deafness; and *4.* the methods available for prevention. One hundred boilermakers were tested using the watch. Their ages ranged from 17 to 67 years, the average being 34.93 years. They had been in this occupation from three to 54 years, the average being 17½ years. For comparison, he took 100 normals, 100 letter carriers and 100 iron-moulders, similarly selected with regard to age and length of time in employment. He found that the letter carriers heard the watch to 79 per cent of the distance

to which it could be heard by the normals, the iron-moulders heard it to 45¾ per cent of this distance, and the boilermakers to 9-1/3 per cent of the distance. In no single instance was a boilermaker able to hear the watch at normal distance. Fifty-nine per cent of them could not hear the whispered voice in either ear. The Rinné test was positive in 90 per cent of the boilermakers, "Pointing," he says, "to nerve structures as the seat of the lesion." Bone-conduction in these 90 per cent was decreased. He stated, "The great preponderance of evidence points to the terminal nerve structures, probably in connection with the basilar membrane of the cochlea, as essentially the seat of the mischief . . . the disturbance began almost immediately after entering upon this kind of work, advancing in many cases slowly, but in some much more rapidly to very decided deafness." He tested 20 of these boilermakers (age not stated) with the Galton whistle and "found the power of perceiving high notes strikingly diminished in every case." Also, "With few exceptions, they reported better hearing in quietness." Habermann,[69] in 1891, reported that he had tested the hearing of 31 boilermakers and found that all were more or less hard-of-hearing. Loud spoken voice was heard relatively better than the whispered voice, bone-conduction and air-conduction for the c(128 d.v.) fork were both diminished, the Rinné test was positive and the hearing for the highest tones was extremely diminished. In 1907[70] he reported another series, this time numbering 107 with similar findings. Cooseman,[38] in 1899, found that the men who ran the beetling machines for working linen were not hard-of-hearing. In these machines, hammers weighing 100 kilos strike the material at a rate of about 100 times per second. He alone of those who have reported studies of this type concludes: "All noisy trades are not necessarily injurious to hearing." He does not report the technique of his tests. Roger,[123] in 1915, wrote: "Although the incidence of noise deafness as affecting blacksmiths, boilermakers, railway and factory employees, engineers and others has long been recognized by the profession and the laymen, no systematic investigation of the subject seems to have been conducted until Dr. Thomas Barr (mentioned above) in 1886 read before the Philosophical Society of Glasgow a paper," etc. Roger was probably induced to start his study by the reports of other European investi-

gators who had become interested in animal experimentation. He tested 48 boilermakers using the 32 d.v., 419 d.v., 512 d.v. and 2048 d.v. tuning forks and the monochord. His conclusions were: "*1.* That loss of hearing for high notes is not, as heretofore taught, the outstanding feature of noise deafness. *2.* That the predominant noises to which the patient has been exposed determined the site of the initial lesion in the inner ear, and that for a considerable time the depreciation in hearing is mainly for sounds of a pitch corresponding to these noises. *3.* That later the unusual vulnerability of the lowest part of the cochlea gives rise to marked loss of hearing for high tones." Roger did not present evidence that the sounds which caused the deafness were analyzed to determine the predominant pitch. He did not use a c^5 (4096 d.v.) tuning fork or the watch in his tests. Had he done so, the results might have been more in agreement with those of Barr and Holt. Lake,[93] in 1923, reported that many of his patients state that they can hear better in noisy surroundings, which is in distinct disagreement with the observation of Barr. He does not indicate the number of patients, but said: "Voice and whisper are very much diminished; Rinné with c(128 d.v.) is positive; bone-conduction is reduced to one-third or less of normal, a totally different picture to that of progressive middle-ear deafness or otosclerosis. . . . What seems to be the course of the affection is, that the auditory nerve is first irritated by the excessive noise, and then gradually injured, eventually beyond repair." Westmacott[151] commented on Dr. Barr's results and concludes, "The mass of evidence favors the view that the principal channel through which the internal ear is injured is through the sound transmitting apparatus of the middle ear and not through other bodily channels." This statement is rather ambiguous, but it is interpreted by the writer as indicating a belief that the trauma was the result of sounds reaching the ear by air-conduction rather than by bone-conduction. McKelvie,[106] in 1933, examined the hearing of 1,011 weavers, using the spoken and whispered voice. He found that 24.3 per cent were hard-of-hearing and that 7.6 per cent suffered from nerve deafness. Vitali,[150] also writing in 1933, says of professional deafness, mentioning boilermakers' deafness specifically: "The acuity for high tones is decreased and bone-conduction is diminished and at times may be absolutely lost. . . The lesions

of the internal ear are produced by air-conduction and the louder the sound and the greater the time of exposure, the greater is the acoustic effect." (Writer's translation.) Audiometric studies of traumatic deafness are almost non-existent. One appeared in an American and the other in an Italian journal in 1935. The Italian article mentions several other studies which were not accessible for this review. Carnevale-Ricci and Bassi's study[32] is described here in some detail because it is not easily available to American students. Those examined consisted of 438 patients selected partly from those presenting themselves at the Industrial Clinic and from out-patient clinics of factories. The effects of advancing age, type and duration of employment, past and present illnesses, urine examinations, blood pressure, the Wassermann test, the use of alcohol and tobacco were carefully considered and a physical examination of the ear, nose and throat was made in each case. The results of the hearing tests of cases with purulent otitis media, with positive Wassermann tests, as well as those with a history of prolonged and excessive use of alcohol and tobacco, whose tests might invalidate the results of the study were eliminated. The audiometric tests of those remaining after these eliminations were classified into three groups: *1.* those in non-noisy occupations, *2.* those in noisy occupations, and *3.* those who worked in an atmosphere of toxic substances, such as lead. These were divided into decades in order to show the effects of advancing age. Their results, in so far as noise is concerned, are arranged in tabular form below.

NOISY OCCUPATIONS.

	Age					
	10 to 19	20 to 29	30 to 39	40 to 49	50 to 59	60 to 69
Per cent having normal hearing	95	58	50	50	22	....
Per cent totally deaf	....	10	18	25	36	90
Per cent with partial deafness, low tone loss	....	10	10	....	8	....
Per cent with partial deafness, high tone loss	5	22	31	25	34	10

NON-NOISY OCCUPATIONS.

Per cent having normal hearing	100	66	60	52	45	15
Per cent totally deaf	....	....	10	10	18	50
Per cent with partial deafness, low tone loss	....	16	15	18	20	50
Per cent with partial deafness, high tone loss	....	18	15	20	17	25

These tables appear rather incomprehensible. It is evident that there is a greater percentage with normal hearing in

each decade in the non-noisy occupations than in those which were noisy, but there seems to be no consistent difference between the various types of deafness in the two groups except perhaps that there is a higher percentage of those with "low tone loss" in the non-noisy group than in the noisy group. This is incompatible with their statement that noisy occupations adversely affect hearing. It is regretted that these writers did not publish examples of the individual audiograms or groups of audiograms for the consideration of the reader and by way of explanation. Is it not possible that some of these cases showed a loss for all tones in the hearing range? Neither is it evident what is meant by "high" and "low" tones, since the frequencies of the stimulus tones of the audiometer are not given, although the article gives the name and maker of the instrument. Beck,[9] in 1927, devised a standard called a "phon" for measuring the noises in industry. He found that "occupations in which there was a noise of 9 phons caused deafness, and that the deafness advances in time and severity in direct proportion to the intensity of the noise." The grinding wheel for sharpening tools made a noise of 10 phons; textile factories, 10 to 11 phons; the revolving drum used in mixing cement, 11 phons; a wire factory, 12 to 13 phons; a boiler factory, 14 phons; and a running locomotive, from 6 to 9 phons.

BOILERMAKING AND SIMILAR INDUSTRIES. AUDIOMETRIC RESULTS.

Fig. 1 shows the audiograms of three cases of nerve type deafness, each with different etiology. The Rinné (with the c^2 weighted Bezold-Edelmann fork) was positive in each case and the loss in acuity for the high tones is much greater than for low. Curve *A* was secured from tests of a physician, age 50 years, whose deafness, according to subsequent tests, is slowly progressive and is probably due to some unknown focus of chronic infection. *B* was secured from tests of a woman, age 67 years, and is interpreted by the writer as being typical of the hearing loss so often seen as an accompaniment of advancing age, the so-called presbycusis. These two curves show a slight loss in acuity for low tones, but this loss gradually increases as the pitch of the stimulus tone rises. In *A* no tones above 4,096 d.v. are heard at the maximum intensity of the audiometer, and in *B* this upper

limit is reached at 8,192 d.v. Curves of this type have been designated by Crowe, *et al.*,[40] as showing a "gradual high tone loss."

C, the left ear of No. 2, Fig. 5, below, is quite different from these two records. The hearing for low tones is essentially normal. Just above 1,024 d.v. there is a sudden sharp drop in the curve, the maximal loss being at 4,096 d.v. In this case there is a history of repeated acoustic insults of various types. The patient is but 30 years of age. These sudden drops in the curves have been designated by Crowe, *et al.*,[40] as being of the "abrupt high tone loss" type. This differentiation between "abrupt" and "gradual" cannot be carried to the extreme, but the terms are quite descriptive. The fact that the curve in *C* turns upward and approaches

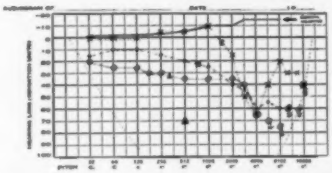

Fig. 1. Audiograms of three patients who have a much greater loss for tones of high pitch than for low tones. (A) is the record of a physician, age 50 years, whose deafness has been progressive for more than four years. (B) is the record of a woman, age 67 years. This is interpreted as being an example of presbycusis. (C) is the record of a man, age 30 years, who has been subjected to loud noises from various sources.

the normal again at 8,192 d.v. is perhaps significant and this tendency will be noted frequently in the curves below.

Audiometer curves presenting the anomaly of an abrupt loss have caused considerable speculation and they have been attributed to a number of causes. Fowler,[55] in 1928, before the American Otological Society under the title, "Marked Deafened Areas in Normal Ears," commented on the frequent appearance of tonal gaps. He stated, "One striking phenomenon seen in a large percentage of hearing graphs is a marked dip in the curve between 1,000 and 5,000 cycles (most often about 3,000 or 4,000). This occurs frequently in both normal and abnormal ears." In 1929,[56] he concluded that normal ears did not show these gaps and discussed the possibility of these gaps resulting from acoustic trauma. He stated, "It would appear that mid-high frequencies were particularly

sensitive to various insults, especially acoustic trauma and toxic neuritis, because they regularly fail to escape in these conditions. . . . It would appear that there is an element of toxic neuritis or trauma diagnosable by the presence of marked deafened areas (dips) and otherwise unascertainable."

This study has forced the conclusion that in a large percentage of cases this sudden abrupt loss must be attributed to the traumatic effects of extremely loud noises. This statement must not be interpreted to indicate that all who are subjected to loud noises will be deafened to the same extent. In fact, the records which follow show that this is not the case. In hazards of any type, individuals will not all be equally affected. Some may suffer severely, while others will escape.

No attempt has been made to secure a large number of cases from any industry for purposes of statistical study. These are records from those who have presented themselves at the Oto-Laryngological Clinic of Washington University, from the private practice of cooperative members of the staff of this department, and a few were examined especially for this study when it was learned that they had been subjected to various types of occupational noises. This study must be classified as a series of case reports, and is consequently open to the criticism which must always await this type of report.

In Fig. 2 and those which follow, each record should be considered by itself and compared with others of this group. All tests were made by the writer in a sound-proof room, using a 1A Western Electric Audiometer. The tentative curve for normal hearing is indicated at the top of each audiogram. The measurement of hearing by bone-conduction for the tone of 512 d.v. is also recorded. (Normal hearing for the bone-conduction receiver used in this study is indicated by a small triangle at 70 s.u. on each chart. This normal should be compared with the recorded threshold on the chart. For example, No. 1, Fig. 2, has a bone-conduction reading of 85 s.u. in the right and 95 s.u. in the left, indicating a loss of 15 s.u. and 25 s.u., respectively, in the right and left ears.) This bone-conduction measurement was checked with that for the c^2 weighted Bezold-Edelmann fork.

The audiograms shown in Fig. 2 are from tests of patients who gave a history of having been engaged in boilermaking or similar industries. The contour of the curves is the striking feature. No. 1 shows a slight loss in acuity for tones

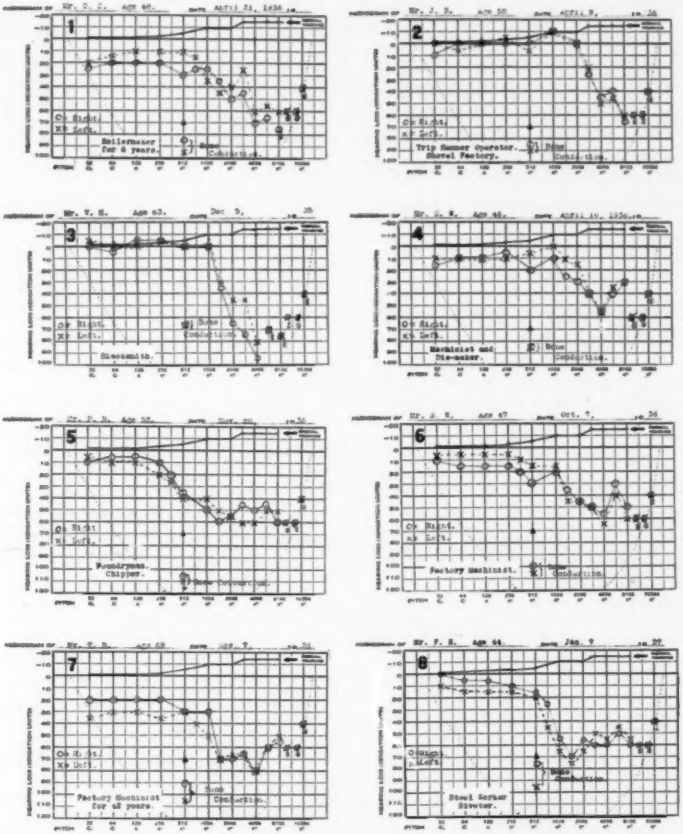

Fig. 2. Audiograms of men who have been engaged in boilermaking or similar industries.

below 1,024 d.v., the loss in the right being more marked than that in the left. At 1,024 d.v. there is a marked drop in the curves for both ears, high tones being very poorly heard and those at the extreme upper limit were not heard

at all at the maximum intensity of the audiometer. (Indicated by arrows pointing downward.) This description, with slight modifications, can be applied to each audiogram in this figure. No. 2 has normal hearing for tones up to 1,024 d.v., and the drop is much sharper than that in No. 1. No. 3 is almost identical to No. 2, except that the sudden drop begins at a slightly lower pitch and is much sharper. In Nos. 4, 6, and 7, the deepest point in the dip is at 4,096 d.v. No. 7 shows a greater loss for low tones than No. 1, but the drop just above 1,024 d.v. is equally sharp. Nos. 4, 6, 7, and 8 show a tendency to return to normal near 8,192 d.v., similar to the curve shown in Fig. 1-*C*.

The histories and clinical notes of these cases are given below:

Case 1: This patient, age 48 years, complained that his right ear had felt "stopped up" for about six weeks. He had been employed as a boilermaker for six years, working both inside and outside of locomotive boilers as a riveter, using a compressed-air hammer. He left this employment 12 years ago. He noted after a day's work his ears would feel stopped up, but his hearing would apparently be all right after a night of rest. He remembered indistinctly that he had earache once when he was nine or ten years of age but cannot tell which ear was involved. Both drum membranes are dull and retracted.

Case 2: This patient, age 58 years, was referred for hearing tests by the Neuro-Surgical department when their examination showed that he could not hear a watch well. He entered the hospital because of dizziness and stated that all of his trouble seemed to be confined to the right side of his head. He had no head noises and prior to the test on the ward, had considered that his hearing was normal. He was a farmer by occupation, but several years ago he had worked in a shovel factory, where his job was to feed the blank metal plates into the trip-hammer for forming. He stated that when the hammer fell on these blanks it sounded like "dynamite." His ears would ring for hours and he would feel deafened after doing this work. This report was conveyed to the referring department as a probable cause of the deafness shown in the audiogram.

The Kahn was negative. The Neuro-Surgical diagnosis was thrombosis of the posterior-inferior cerebellar artery, right.

Case 3: Six weeks before this test was made, this patient, a man, age 63 years, was knocked down by a street car, striking the right side of his head on the pavement. He was dazed by the accident but not unconscious. He claims that he bled from his right ear, nose and mouth. He was placed in a hospital, where he was in bed for three weeks and, when taken home, remained in bed for 10 days more. He did not vomit. He felt slightly nauseated, and when he lifted his head was somewhat dizzy. He states that the hearing in the left ear is and has been good at all times, but the right ear feels stopped up and the hearing in the right ear, which had been good before the accident, is now very poor. He is a blacksmith by trade and has followed this occupation all his life.

The fact that the hearing in the right ear is almost equal to that in the left raises the question as to the status of the hearing in the

right before the accident. If it is assumed that the accident caused this deafness in the right ear, it appears probable that it was also responsible for the deafness in the left. On the other hand, blacksmithing has so long been known to cause deafness, it cannot be denied without the evidence of hearing tests before the accident that the hearing in both ears may have been defective as a direct result of his occupation.

Case 4: This is the record of a machinist and diemaker, age 46 years. Four months before the date of this test, while the patient was grinding a metal bar or plate with a high-speed emery wheel, the wheel broke and a fragment struck the left side of his head and knocked him unconscious. He recovered in a hospital, where he remained for five weeks. He did not vomit. He complained that he was slightly dizzy when he was able to be up and is still dizzy and staggers a little when he walks and especially when he looks upward. He has head noises like the sound of crickets. To declare that his hearing loss was the result of skull injury, one must assume similar damage to both ears, for the hearing is diminished almost equally in both. On the other hand, it cannot be definitely stated without the evidence of tests, that the deafness was not the result of the extreme noises, such as that made by grinding with a high-speed emery wheel. The fact that the hearing loss is bilateral and similar to others of this group is strong evidence that it resulted from the noises of his occupation rather than from the head injury. The tympanic membranes were intact, the right being slightly retracted.

Case 5 is the record of a foundryman, age 52 years. A native of Bulgaria, he served three years in the Bulgarian cavalry during peace time and did no shooting at all. He came to America and has been employed in one foundry for 28 years. His wife states that when he entered this occupation his hearing was good, but she noted after he had worked for four years, his hearing was less acute. His specific work is called "chipper;" i.e., with a compressed-air chisel he chips off the rough spots on castings as they come from the moulds. His wife states that she knows at least 20 of his fellow employees who are hard-of-hearing. These men always wear large packs of cotton in their ears while at work. The Kahn was negative. The tympanic membranes were moderately retracted.

Case 6 is the record of a man, age 47 years, who has been a machinist since 1911. During this time he did general machine and repair work in several factories. It usually was quite noisy, but the noises were never of an explosive nature. He thinks that his deafness has progressed somewhat during the last four years. He was in the U. S. Infantry for eight months during the World War, but he did not see foreign service and during the time of his enlistment he did no shooting. He remembers that he had earache when he was a child, but is quite sure that his ears did not discharge and does not remember which ear was involved. Both drum membranes are somewhat retracted.

Case 7 is the record of a machinist, age 68 years. Before 1929 he worked in several very noisy machine shops for 42 years. Twelve years ago he went to an otologist because he noted some hearing loss. This otologist removed some impacted cerumen and the patient thought his hearing was improved. He thinks that his deafness has not progressed during the last three or four years. The drum membranes are retracted and dull.

Case 8 is the record of a structural steel worker, age 44 years. Fifteen years ago he was employed in a steel plant, riveting steel plates for the construction of coal hoppers, and frankly attributes his deafness to the noise of this occupation. He is still employed in steel construction work, but because of the noise, has changed his work and

now heats the rivets for those who do the actual riveting. He has little difficulty in understanding the conversation of his fellow workmen, for they all speak loudly, but he notices his deafness when he uses the telephone and in conversation with his family at home. He has continuous tinnitus which sounds like a "peanut whistle." He has done no shooting. He denies earache, discharge and dizziness. The drum membranes are dull in appearance, are thickened and the ossicles move well with the Siegle's otoscope.

DEAFNESS FROM GUNFIRE—
EVIDENCE FROM THE LITERATURE.

The *Lancet* for Aug. 4, 1866,[95] printed the following query by one who signed himself, "Volunteer":

"Can any of your readers suggest a remedy for preventing deafness caused by rifle shooting?"

This was answered in a later issue by John Chippendale, M.R.S.C., as follows:

"In answer to the query of 'Volunteer' in your last issue, allow me to suggest the wearing of cotton in the ears while at rifle practice. This is done, in France at any rate, by artillerists to prevent deafness and bleeding of the ears."

This proves that the noise of gunfire was recognized as a cause of deafness in 1866, and that some at least had found a method of prevention. That deafness might be produced by the explosion of guns smaller than those used in the artillery, we have the evidence of Bruner,[25] who in 1880, reported a case of deafness in a person who was in a room when a single shot was fired near one of the windows. The deafness was very marked immediately after the shot was fired. Later there was some improvement, but after two years, a watch which was normally heard to 500 cm. was heard to 200 cm. by the patient. The Rinné test was positive. Wolf[156] reported a similar case in 1874. Sexton,[133] in 1888, described the trauma in the tympanic membranes of soldiers who were near a large shell when it accidentally exploded. Nimier,[109] in 1889, stated that much of the deleterious effects of explosions on hearing were not due to the trauma produced in the middle-ear but resulted from the effects on the nervous apparatus of the cochlea. Noel,[108] in 1891, agreed with Nimier and thought the condition in the ear was similar to that occurring in the eye when it was subjected to strong luminous stimulation. Castex,[33] after exam-

ining the middle-ears of two persons who were killed in an explosion and examining and testing the hearing of three other persons who were in the same accident but escaped death, noted perforations in the posterior half of the drum membranes but could discover no lasting effects on the internal ear. In 1899, Mueller[104] examined the ears of 96 soldiers who were in the artillery. Forty-four ears showed vascular congestion and there were extravasations of blood in seven, but in no case was there a rupture of the tympanic membrane. Bone-conduction was diminished. Cases of injury to the ear resulting from the firing of large guns during the Spanish-American War were reported by Emerson,[50] Kipp,[88] Dench,[45] Simons,[138] Duane,[48] and Lewis.[97]. Jürgens,[85] in 1907, examined 13 cases of injury to the ear following the explosion of bombs. He found the main symptom was a loss of hearing for low tones and these showed a tendency to recovery after a short time. The semi-circular canals were unaffected. McMaster,[107] in 1909, reported that of 50 men who were in the British Navy, more than half were practically deaf, the deafness being attributed to the action of the larger guns. Hofar,[75] in 1913, reported that the most marked effect in cases of hearing loss due to gunfire was a loss for tones of high pitch, but he did not think this could be attributed to the effects of the explosive noises. Horne,[77] in 1914, noted the deafness that comes in sportsmen who use small arms, and pointed out the fact that a locomotive whistle in a tunnel could cause deafness. He said, "The injurious effects of the shock of concussion are upon the terminal portion of the nerve structure of the ear." Love,[100] in 1916, stated that he "feared that great slackness had existed" in the examination of recruits. He advocated the use of the whispered voice as a standard in these tests. Soon after the beginning of the World War, many British writers described the conditions found in the ears of soldiers who had been deafened in the service. Got,[61] in 1916, stated: "The deafness is always of the labyrinthine type . . . there is abolition of cranial perception to a watch, abolition or considerable diminution of aerial perception, diminution of acuity more marked for high than for low sounds, for whisper than for spoken voice. Rinné positive or barely negative; lateralization of the Weber test to the sound or less affected side . . . if their deafness increases it is on account of some intercurrent cause arising." Bryant,[27] in 1917, classified the effects of explosive sounds into three cate-

gories: *1.* Those in which there is a rupture of the tympanic membrane; *2.* those with complications following the rupture of the tympanic membrane; and *3.* commotion of the VIIIth cranial nerve apparatus. He stated that the cochlear branches always suffer and the vestibular branches less frequently. Trible,[145] in 1917, described the auditory acuity requirements for men in the naval forces of the various powers. Jobson,[82] in 1917, tested the hearing of 73 soldiers who had had at least four months of exposure to the noise of gunfire. The c^2 fork was selected for the tests because "the higher forks are heard for such short periods that no accurate results could be obtained in an investigation of this kind." Sixty of the 73 had no otitis media. While many of them gave a definite history of severe deafness after a day or two of exposure, at the time of the tests 80 per cent of them were not aware of any deafness, but the c^2 fork showed deafness for both bone-conduction and air-conduction. Wilson,[154] in 1917, found diminished hearing for all forks after exposure to gunfire, but thought the prognosis for recovery "good as a rule, especially where there was no trauma demonstrable in the peripheral organ." He divided the cases of deafness after exposure to the noise of gunfire into three groups: *1.* Cases of nerve deafness; *2.* cases with nerve deafness and a fixed idea that they cannot hear; and *3.* malingerers. He stated, "The pathology of nerve deafness from high explosives is unknown." In 1918,[155] he stated that there was diminution for all tones by air-conduction and bone-conduction, that the loss was least marked for c^2 (512 d.v.) and c^3 (1,024 d.v.) and when almost completely deaf there might still be a remnant of hearing by bone-conduction for tones near 512 d.v. Yearsley,[158] in 1917, noted that this type of deafness might be quite severe at first but that the hearing for the whispered voice improved slowly. Guild,[67] in 1918, studied the efficiency of the various types of devices used in warfare for protecting the ears of gunners. He at first used animals in his experiments, but later had a satisfactory mechanical device. He also prepared[68] an extensive bibliography of 140 writers, not only of those who were interested in animal experiments but also of those who were interested in the clinical side. In his review he stated, "It is evident that gun-deafness has long been recognized by those nations using fire-arms of various types." As a result of his experimental observations, he found that the closure

of the external canals with packs of cotton thoroughly impregnated with vaseline was the most effective means then known for protecting the ears from the traumatic effects of gunfire. Shuter,[137] in 1918, wrote that he suspected the conducting apparatus, including the perilymph and endolymph, was the part wholly affected in concussion deafness. The description by Loeb,[99] in 1918, is fairly comprehensive. He wrote concerning "Indirect Trauma" as follows: "Under this head are grouped those cases which result from traumatism propagated from adjacent structures from the explosion of shells in the neighborhood and from the constant detonations to which soldiers, especially artillerymen, are constantly exposed. This comprises an exceedingly large group of war accidents which, in part at least, are comparable to certain occupational concussions in time of peace." The symptoms are, according to him: "Deafness either complete or less marked is always present, it may remain permanently or it may continue for any period between these two extremes. The deafness is of definite labyrinthine type, with abolition or marked reduction of bone-conduction, diminution of auditory acuity, more marked for high than for low pitched tones, Rinné positive, Weber lateralized to the sound or less diseased side. . . . Simple labyrinthine concussion, as a rule, recovers in a few weeks, the milder cases without any loss in hearing. The severer cases may recover from all symptoms except deafness, which remains unchanged or increases. In like manner, the cases due to continuous noise from artillery, etc., may remain deaf." Ingersoll,[79] in a report of his work with the A. E. F. which was read before the American Otological Society, stated that there were 30 cases of bilateral deafness among the soldiers due to the concussion of exploding shells. These were all seen by him at the U. S. General Hospital for head surgery. He stated, "In some cases the drum-membranes were ruptured by the concussion, but in many of them there was, at least, no evidence, and no history, of ruptured membranes . . . the deafness which was complete immediately after the concussion almost invariably improved somewhat, so that the men could hear a loud voice from one to two feet, and were able to hear their own voice and modulate it." According to Ingersoll, there were 106 cases of bilateral deafness in the A. E. F. from the beginning of activities up to March 1, 1919, all of whom were sent to the same hospital. In

about 30 per cent of these the deafness resulted from shell explosions. Richardson[119] also described the deafness occurring in the A. E. F. He stated, "There is a large group of cases, properly designated as concussion deafness . . . which are attended with marked improvement to complete loss of hearing. Very few of these ears show any tendency to improvement or to restoration of their hearing. They are most due to the prolonged exposure to highly explosive shells." Colledge[36] described the condition as follows: "The lesions from gunfire showed a certain variety: First, those in which the deafness was gradually progressive from prolonged exposure to the noise of gunfire; and, second, those in which the deafness resulted from a single blast, without in either case any visible change but with definite loss by bone-conduction. In some cases of deafness following a single explosion, there was not only definite loss of bone-conduction, but, on inspection, the tympanic membrane was seen to be dotted with punctate hemorrhages. Sudden deafness was more likely to be caused by a bursting shell or exploding mine than by the actual firing of guns. There is also a class of case in which there is actual rupture of the tympanic membrane." Dickie[46] made quite a similar classification. Faulder[52] reported that in 20 years of service in the artillery of the British Army, including three years in the World War, he had in but one case found the tympanic membrane ruptured as the result of the firing of a gun. Grimwade[65] noted the frequent appearance of middle ear lesions among naval personnel. Of 167 men whom he examined, 74 per cent were suffering from middle ear disease and 3 per cent had deafness which was "definitely attributable to the service—namely, traumatic rupture and labyrinthine deafness." In summarizing the work of the Otological Service of the U. S. during the World War, the report of the Medical Department[117] stated, "Concussion deafness. This was the distinctive military type of deafness. It was usually the product of actual warfare and its appearance was largely confined to our troops in France." It further states, "Treatment is of little benefit, and a transfer to some other branch of the service is usually advocated in order not to add insult to an injured organ." Michie[101] advocated audiometric tests to determine the amount of disability arising from the deafness in officers who were retired from the U. S. Army. He found that 10.415 per 1000 of 778 officers who were retired between November,

1918, and the fall of 1923 had "defective hearing as a direct or contributing cause." The men were divided among the various services as follows: In the aviation service, 17.88 per 1000; infantry, 39.09 per 1000; and cavalry, 39.31 per 1000. No mention is made of retirements from the artillery where, from the reports of others, the greatest percentage of deafness should occur. Ridout[118] made audiometric tests of 225 naval officers from 20 to 60 years of age and found increasing loss for tones of high pitch in each successive decade, the low tones being relatively unaffected. This high tone loss he attributed to the effects of gunfire. Dore[47] pointed out the frequent occurrence of deafness among hunters. He found it more marked in the ear which was held closer to the muzzle of the gun, *i.e.*, in the left ear of a hunter who shot right-handed, and vice versa. This deafness he found was most marked for c^3, c^4 and c^5 forks, and varied with the amount of the trauma. Bone-conduction was decreased and the tuning forks in the Weber test were lateralized to the unaffected side. This, according to Dore, clearly localized the lesion in the acoustic labyrinth and indicates that the deafness is similar to all other deafness resulting from excessive sound stimuli.

DEAFNESS FROM GUNFIRE—AUDIOMETRIC STUDIES.

A.—LARGE GUNS.

Fig. 3 shows the records secured from tests of men who, according to their histories, have been subjected to the noise of guns of large calibre. The selection of cases was limited to those presenting themselves at the Clinic, those from colleagues who kindly referred them for tests, and those who were tested in the search for material for this report when it was learned that they had had service in the artillery. One (No. 7) had a history of injury and deafness from the effects of the shell exploding close to him. No. 2 was a naval officer whose retirement for disability because of his deafness was being considered by his superiors.

The contour of the curves is strikingly similar to those shown in Fig. 2. The peculiar feature in nearly all was the relatively good hearing for the tones in the lower part of the range of the voice (from about 300 d.v. to 3,000 d.v.) and ability to hear spoken words well. The audiograms, ex-

cept No. 7, show normal hearing or very slight loss for tones up to and including 1,024 d.v. For tones above this pitch there is consistently a sudden, sharp bilateral loss, higher tones being heard very poorly or not at all. In No. 3 the loss

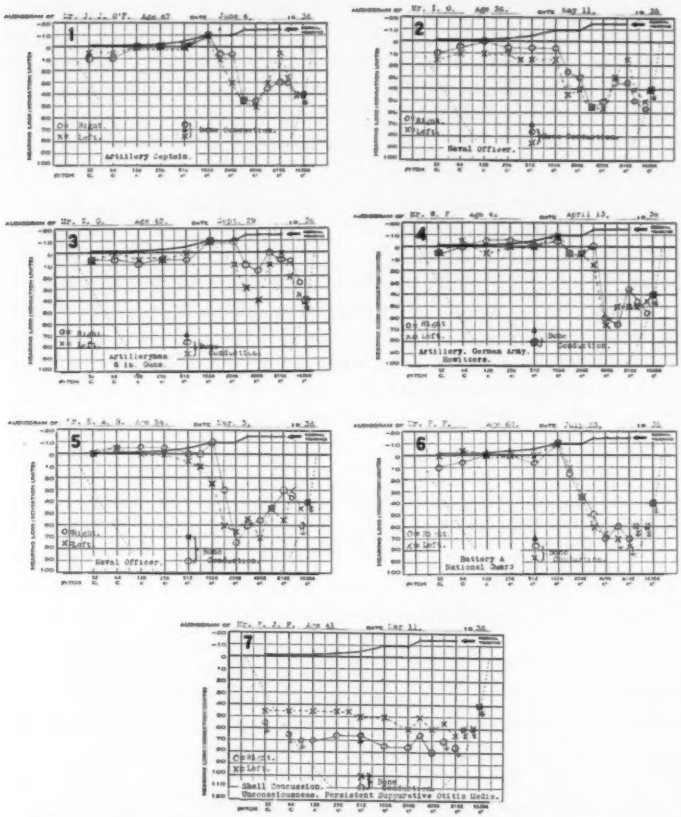

Fig. 3. Audiograms of soldiers and sailors who have been exposed to the effects of the noise of large guns.

is slight and is characterized by a dip in the curves for each ear, most marked at 4,096 d.v. and deeper in the left than in the right. This man was at the front for but two months, according to his statement, and the amount of noise to which he was exposed during this time is a matter of conjecture.

This is similar to many of the records shown in Fig. 4. In No. 5 there is a history of but a single shot, but as a naval officer he must have been subjected to gunfire at other times.

The histories and clinical notes are given below:

Case 1: This patient, age 47 years, was an artillery captain during the World War, in charge of four 155 mm. guns for 22 months. His quarters were located in the centre of the placement of these guns. He now notes a very slight feeling of fullness in his ears and a slight loss of hearing which he quite frankly attributes to the noise of gunfire. He stated that he thought 90 per cent of the men who served under him were hard-of-hearing from the same cause, although the men were supposed to wear cotton plugs in their ears when firing. The patient used the Elliott protectors.

At 2,048 d.v. (c^4) the hearing in the right ear is almost normal, while that in the left is down rather markedly. The greatest loss is shown at 4,096 d.v. The hearing in both ears tends to return to normal at 8,192 d.v. This deafness could not have been ascertained in tests with a set of tuning forks which used the c^4 fork for the upper limit. Case 3 and Case 4 show this characteristic even more noticeably than Case 1.

Case 2: This record shows the hearing of a 35-year-old naval officer, in active service. He was in immediate command of the men who manned the guns in the turret of an American battleship. Because of his defective hearing, his superior officers were considering that he be recommended for discharge. Since these curves are similar to the others shown in this study, the opinion was expressed that his hearing loss was probably the result of the noises of gunfire and if his discharge entailed any loss to him it would be unjust, because it would penalize him for the performance of his duties.

Case 3: This man, age 42 years, was in the hospital in the service of the Medical Department. He was tested in the search for material for this study simply because he gave a history of having been in the American artillery during the World War. He was not aware of any hearing loss. He was one of a squad who manned a 6-inch gun. He noted after firing that he would feel deafened, but this sensation would pass off after a few hours. He was in active duty at the front for only two months. The number of engagements in which he served is not known. He used no protective device and received no instruction as to how he could protect his ears. He is a printer by trade. The medical diagnosis was colitis. The Kahn was negative.

Case 4: This man, aged 41 years, was tested in the search for material for this report. He was not a patient in the hospital, nor did it appear that he was hard-of-hearing. He was tested because it was known that he had been in the infantry and the artillery of the German army during the World War. He is the instrument maker for this department. During his army service, because of his mechanical ability, he was in charge of the aiming mechanism of one of the 35 cm. Howitzers. He states that the gunmen were instructed to hold their mouths open when firing the large guns. Later, in the infantry, he often fired his rifle so rapidly that the barrel became too hot to hold. After both types of firing his ears would ring and he would feel deafened for some time, but these sensations would disappear in a short time. During the last year he discovered that he was not able to hear the chirping of crickets as well as others, but he has noted no other deafness.

Case 5: This patient, age 54 years, noted his hearing loss in the fall of 1911, while he was an executive officer on board an American battle-

ship. One day he came from his quarters and passed close to one of the smaller guns on deck just as it was fired by his men. He was quite deaf as a result and immediately consulted an otologist in New York City. This otologist told him that his hearing for high tones had been damaged and that the resulting deafness would probably be permanent. Tests repeated March 3 and on Sept. 24, 1936, showed the hearing had not changed during that time. He is now a bank executive and notes some loss of hearing for conversation and in the use of the telephone.

Case 6: This patient, age 62 years, is one of the oldest in this group and the probable effects of advancing age must be taken into consideration in accounting for his hearing loss. Up to the age of 20 years he had been a carpenter. He then studied law and has been practicing since that time. He complains that his head noises make it difficult for him to understand conversation. These head noises have been prominent for at least 35 years. From the time he was 12 years old he has done a great deal of hunting and shooting, but he did not notice that this affected his hearing. When he reached maturity, he enlisted in Battery A of the National Guard. While in this service he was frequently on the range firing a 3-inch gun. After target practice he would notice that the tinnitus would be much more annoying and his hearing would be poorer for some time. He was discharged from the National Guard in 1911.

The drum membranes are gray and have a fair light reflex. Both are retracted and the short processes stand out rather prominently.

Case 7: Male, age 41 years. The records in this case are quite similar to that shown in the right ear of Fig. 9, Case 2. In August, 1918, while the patient was a soldier in the A. E. F., a shell exploded close to him while he was in a trench. He recovered consciousness the following day in a field hospital. Both ears were discharging and he seemed to be totally deaf. Under treatment, the otorrhoea continued for five months while he remained in the army. The hearing gradually improved, but never became normal. These circumstances make it impossible to estimate the loss of hearing due to acoustic insult alone. The chronic suppuration which followed the rupture of the tympanic membranes would, in the course of time, doubtless have a deleterious effect. If the discharge ceased, the fibrous and adhesive processes which sometimes form in the middle-ear might, in the course of 18 years, cause the deafness to progress. He stated that he thought his hearing had been getting poorer for the last five or six years.

B.—SMALL GUNS.

Fig. 4 shows the records of nine men who gave a history of having been exposed to the noise of the explosion of small arms or similar explosive noises. Nos. 1, 2, 3, 5, and 8 were tested in the search for material for this report and were not aware of any hearing loss at all. This is not difficult to understand for the loss in nearly every case is above the range of the spoken voice. Tests using the spoken voice alone would not reveal any hearing defect except possibly in Nos. 7 and 9. Tests with a set of tuning forks which did not contain a c^5 (4,096 d.v.) fork would likewise reveal losses in but a very few.

Theoretically, it appears that the weakest explosive noise sufficient to produce trauma caused a decrease in acuity for

tones near 4,096 d.v., as in Nos. 1, 2, 3, 4, and 5. With continued or greater insult to the hearing mechanism, this gap

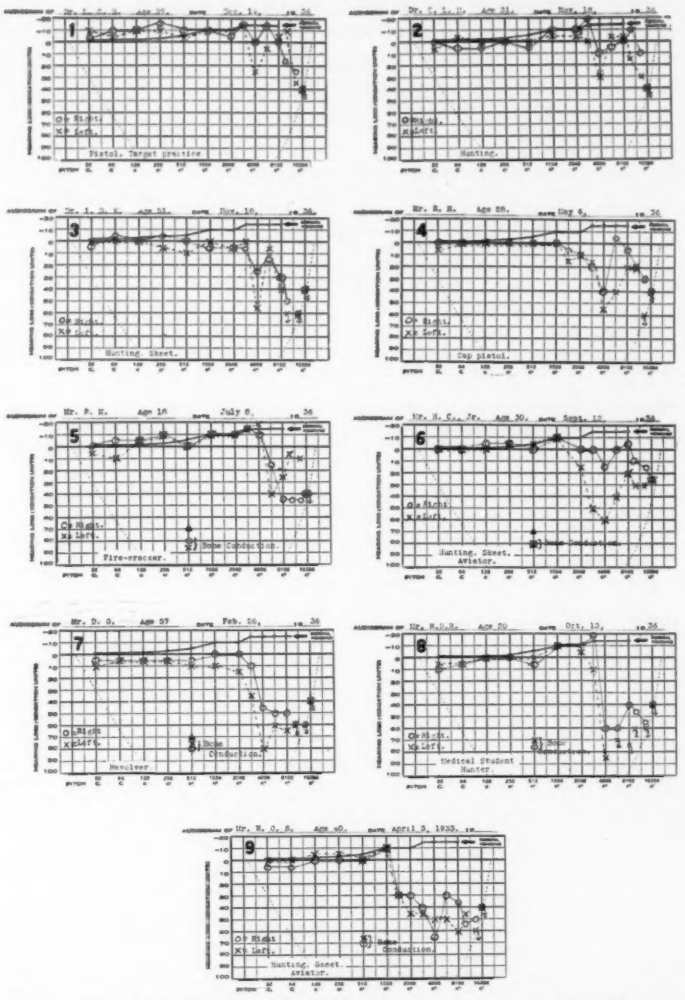

Fig. 4. Records of men who have been exposed to the noise of small guns.

at 4,096 d.v. broadens in both directions, as in No. 6, and ultimately the defect causes a loss in hearing for the ex-

tremely high tones, as in Nos. 7, 8, and 9. The low tones are relatively unaffected.

This loss cannot be attributed to the effects of advancing age for No. 8, the record of a 20-year-old medical student, shows a much greater loss than that in No. 3, the record of a physician 51 years of age.

Case 1 is the record of one of the staff members of this department. He casually mentioned that one afternoon recently he went to the police pistol range (see also Fig. 5, Cases 7 and 8) and did some target shooting. He denied that it had affected his hearing, but because of his interest in this study, he consented to be tested. The loss in the left ear at 4,096 d.v. is quite definite. He can, by interchanging the receiver, hear this tone distinctly with the right ear when it is inaudible in the left. He is right-handed.

Case 2: This record shows the results of tests of an interne in the hospital. He had done some hunting and trap-shooting during the past few years and was tested in the search for material for this study. He has noticed a feeling of fullness in his ears after he has been shooting. He has found that vaseline impregnated plugs of cotton in the ears will prevent this sensation. He denies all previous ear trouble and even now notes no deafness. The dip in both ears, especially in the left, at 4,096 d.v., is the striking feature of the record. He is right-handed.

Case 3 is the record of a physician, a member of the staff of this department, age 51 years. He has never noted any deafness and with some reluctance consented to be tested in the search for material for this study. It was known that he has done considerable hunting and shooting. In this record, the dip at 4,096 d.v. is more marked in the left ear than in the right. He shoots right-handed. Both this record and Cases 1 and 2 show an additional loss for tones at the extreme upper range of hearing. Losses for the very high tones are encountered in tests in nearly all persons who reach mature age. The cause is not understood. It has been considered that they are an accompaniment of advancing age, but this is a statement of fact rather than an explanation. In these three records the losses for the extremely high tones is greater in the older physician.

The drum membranes are normal except for slight retraction, possibly more marked on the right. There had been a middle-ear abscess from a blow on the ear from boxing, but there is no visible scar. There is also a history of traumatic rupture of the left membrane, followed by intense vertigo and nystagmus, with serous discharge for several days, as a result of diving. Just what part each successive trauma played in causing this slight deafness is not possible to estimate. Each might have caused losses greater than are shown here.

Case 4: This patient, age 28 years, was playing with his small nephew who was flourishing a cap pistol. The child approached his uncle from behind and to the left, and fired the pistol at a distance of six or eight inches from his head. The patient noticed deafness and tinnitus immediately, and came to the clinic for treatment. The tests were made one month after the accident. He was a shoe-pattern grader by trade, an occupation which is not noisy. He has been hunting a few times in his life, always using a 22-calibre rifle. He has fired a shotgun just once.

Although no tests were made before the accident, in this case we have probably the most direct sequence of events of any of the cases

presented. He was first tested three weeks earlier by one of the junior staff members, who referred him for this test. The two tests show almost identical results, indicating that there has been no change in that time.

The drum membranes were somewhat retracted and slightly dull, but moved normally with the Siegle otoscope. The Kahn test was negative.

Case 5 shows the record of a high school boy, age 18 years, an amateur radio operator, who recently enlisted in the U. S. Navy as radio operator and was waiting to be called into service. On July 4, he lighted a 3-inch firecracker and as he drew back his right arm to throw it, it exploded directly in front of his ear. He was deafened by the explosion, his ears rang, and since that time he has had a feeling of pressure in his ears. He came to the clinic on July 8 to find out if anything could be done to improve his hearing, for he feared that the deafness might bar him from the contemplated naval service. Tests repeated three weeks and six months later showed the same hearing loss. He passed the whisper test required for naval service and was accepted.

This case also has a definite and immediate sequence of events, showing the cause and effect relationship between deafness and acoustic trauma. But even here we have no definite knowledge that the dip in the curves just above 4,096 d.v. did not exist before the accident. The fact that he was a "ham radio operator" and had noticed no hearing loss before is distinct, but not definite evidence that his hearing was normal before the accident.

The drum membranes were normal.

Case 6: This patient, age 30 years, has done a great deal of shooting and hunting. Four or five years ago a shotgun was accidentally discharged quite close to his head. He has also done considerable flying in a two-seater open cockpit plane and he notes that his hearing will be poor for some time after he has been flying. Which of these two probable causes is responsible for his hearing loss is impossible to state. He is right-handed, however, and since the defect is more marked in the left ear, it seems that shooting rather than flying is more likely the cause.

There is a history of a great deal of deafness in his family, but he has never noted any hearing loss. He was referred for tests by a colleague to whom he appealed for advice. He was planning to marry and wished to know if there was any evidence that he might transmit this familial deafness. He was questioned concerning his occupation before these tests were made.

The tympanic membranes are normal and there is no history or evidence of ear suppuration.

Case 7 is the record of an old Texan, age 57 years. He denied all ear trouble except earache in childhood which he remembers indistinctly. He has done some flying, but has not noticed that his hearing was affected in any way as a result. When he was a young man, one favorite sport of those in his gang was to come unexpectedly behind a companion and fire a revolver close to his head to see how far he would jump. Not only has he indulged in this sport, but he has had the surprise reciprocated. He has done a great deal of shooting, especially with his six-shooter. He is right-handed.

The Wassermann and Kahn tests were negative. The tympanic membranes were somewhat retracted.

Case 8 is the record of a medical student, age 20 years, a native of Wyoming, who was found to have defective hearing for the watch in

his routine physical examination. He noted difficulty in hearing conversation when other noises interfere but has no difficulty in hearing his class-room instructors. He has been attending school practically all his life and in vacations has clerked in a drug store. He states that he goes hunting at every opportunity. He is right-handed.

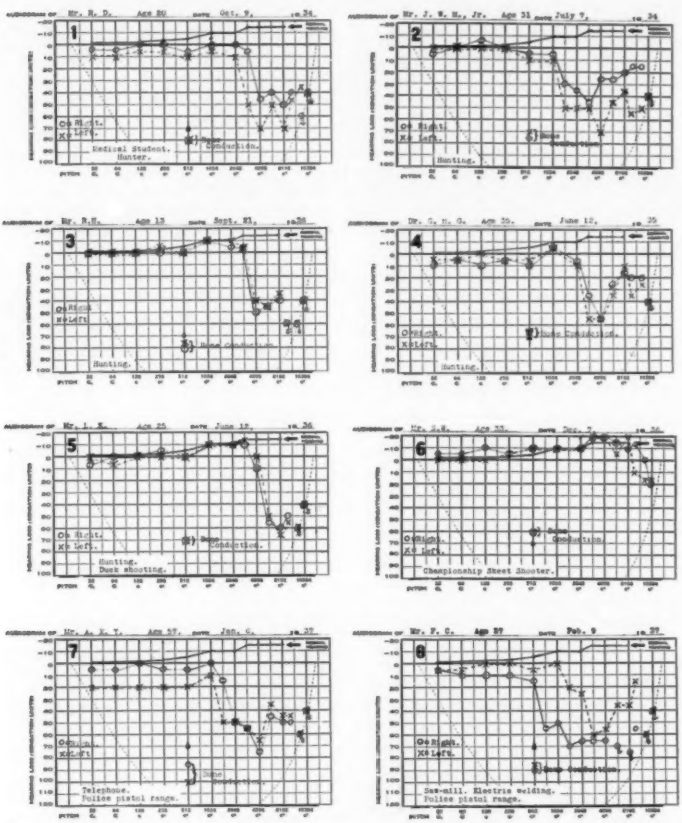

Fig. 5. Records of men who have been exposed to the noise of small guns.

Case 9: This patient, age 40 years, was in the U. S. Aviation service. He often noted that his hearing would not be good for a time after he had been flying. In addition to this, he has done considerable shooting, both at skeet and in hunting. During the last four years he has done more hunting than usual. Tests made in 1936 show that the deafness has not progressed during the last three years.

Both tympanic membranes are normal. There is no history of suppuration.

Within the last few weeks he has had an abscessed molar tooth extracted. Since then, joint pains from which he has been suffering for several years have subsided. The question arises as to whether this deafness might have resulted from this toxic focus rather than from trauma. The fact that the deafness has not progressed for three years and that the audiometer curves show a sudden marked drop instead of a uniform loss leads to the assumption that this is the result of trauma. Whether the deafness resulted from the noises of his airplane or from shooting is problematic. It is included in this group because it is similar in contour to others in the group who have been subjected to the noise of gunfire (see also Fig. 4, No. 6).

Case 1 is the record of another medical student, age 20 years, who was found to have defective hearing for the watch during his routine physical examination. He had earache in one ear when he was four years of age, but does not remember which ear was affected. He notices difficulty in hearing conversation when it is faint and cannot hear well through the telephone when it is located in a noisy place, defects which are common to everybody. He has no difficulty in hearing his instructors. In the past he has done a great deal of hunting and noted that the noise of gunfire made his ears ring. When he was about 14 years of age, he was returning one day from a hunting trip in his automobile. His shotgun, which was resting in the car at his right with the muzzle pointing upward, was discharged when the car passed over a bump. The charges from both barrels passed through the top of the car. He was quite deaf for several days after the accident, his ears rang a great deal, but he thought that his hearing had returned to normal.

Case 2: This patient, age 31 years, has done a great deal of hunting. Often when sport was good he would shoot until the gun-barrel was too hot to hold. Frequently after shooting he would note considerable ringing in his ears. Since 1934 he has done no shooting at all. Tests made in 1935 and 1936 show that the deafness has not progressed. He is right-handed.

The right drum membrane is normal in appearance; the left is slightly thickened. Both move normally.

Case 3 is the record of a schoolboy, age 13 years. His parents and an otologist in a neighboring city had discovered that he did not hear high tones well. They suspected some toxic focus as the etiological factor. When the sharp dip at 4,096 d.v. was discovered in the audiometer test, trauma was suspected and the boy was questioned. He had done considerable hunting with a 22 rifle. It was thought that guns of this calibre fired in the open do not make sufficient noise to cause deafness, so he was questioned further. He is right-handed and should the deafness have been caused by his own gun, the loss should have been greater in his left ear. Further questioning brought out another incident. A year prior to the date of this test, his brother fired a shot-gun just to the right and above this boy's head. He was deafened by the noise, his ears rang for the remainder of the day, but the hearing seemed to be all right the following day and he did not mention the incident to his parents.

Histories are not always dependable. It seems probable that had such an accident occurred to anyone, it might soon be forgotten. In this case the sensation was so unpleasant that he remembered it distinctly. The brother and this boy secured a shot-gun and a box of shells and fired them all in an afternoon.

The right drum membrane is normal. The left is retracted in the posterior superior quadrant, but there was no evidence of infection about the nose, throat or ears when this examination was made.

Case 4 is the record of a physician, age 35 years, who in the past has done a great deal of hunting and trap-shooting. He first noted some

loss in hearing after he had been trap-shooting. He is right-handed and has noted that the hearing in the right ear is better than that in his left.

Case 5: A man, age 25 years, in the Neuro-Surgical department, was referred for hearing tests when it was found that he did not hear a watch well. Up to this time he had considered his hearing normal. The sudden drop in the curves at 5,793 d.v. and 8,192 d.v. was ascertained after his history was secured and aroused suspicion that the loss might have originated in some acoustic insult. He was questioned further about his occupation. He was a draughtsman who for recreation did a good deal of duck shooting. After shooting, his ears would feel "stopped up" and would ring for some time.

The Neuro-Surgical diagnosis was medullablastoma of the fourth ventricle. The blood Kahn was negative.

Case 6: One of the members of the staff of the department who was interested in the problem of gun-shot deafness referred this man for hearing tests. He is 33 years of age, a champion skeet-shooter who has recently won a skeet tournament. He denied any hearing loss and the record is one of the best ever secured in tests of a person of his age with the audiometer. It is safe to say that his hearing is normal. Skeet-shooters in tournaments often will fire 150 rounds in an afternoon. Several contestants stand in line a few feet apart, each firing in turn at the flying targets. Each is exposed not only to the noise of his own gun, but also to that of his rivals, and if gunfire causes deafness, these shooters would be hard-of-hearing. This man explained that when he first practiced shooting, the effect was so annoying that he has always worn heavy cotton plugs in his ears when he is shooting.

This example may be an excellent argument for the use of some protective device when shooting. On the other hand, all the hazards of life affect individuals differently. It may be possible that through some natural means his ears are not affected as are those of others similarly exposed.

Case 7 is the record of a policeman, age 57 years, who has been on the force for 23 years. In 1930, while using a telephone at his left ear, he received a severe electric shock through the receiver (see also Fig. 6). He was knocked to his knees but was not unconscious. Witnesses reported that a flash of fire came from the receiver. He was informed that men were working on the telephone line at the time. This accident occurred in January, when the streets were covered with ice and snow, so it seems probable that this high voltage on the telephone line did not come from a bolt of lightning. He was deafened by the shock, his ears rang and still continue to ring. He has recently been told by the police physician that he will become totally deaf within a year.

It was easy to tell in conversation that this man was hard-of-hearing in both ears. The telephone accident did not account for a bilateral loss, so he was questioned further. Twice each year the men on the force must pass a test in marksmanship. Forty shots are fired with a special 38 calibre revolver. If a certain standard is not met, they must repeat the test. This man had been shooting on this range within the past week and said that with each shot it felt as if something were being thrust into his ears. They felt stopped up for two days afterwards. Early in his service he had been a crack shot and a member of the revolver team. He failed in his last test because the unpleasant sensation of the shooting made him nervous.

This revolver range is described as being located in a room approximately 80 ft. by 40 ft., with a 12-ft. ceiling. The targets are located at one end of the room and the marksmen, when shooting, stand in small

booths at the opposite end (see also No. 1, Fig. 4). Operators of commercial shooting galleries know that the shooting of even small calibre guns must be done in the open and the marksmen usually stand at the open end of the gallery. It seems that the shooting gallery provided for these policemen is poorly designed for the protection of the hearing of the men.

Just what part the telephone accident and the revolver practice played in the production of this deafness is a matter of conjecture, since there were no previous hearing tests. If it is assumed that the revolver practice is responsible for the deafness in the right ear, it must have caused similar effects in the left. He had been practicing in the gallery for years before the telephone accident took place. Both ears show equal losses for high tones. The left ear shows an additional loss for low tones. Can we assume that this low tone loss represents the effects of the telephone accident (see also No. 5, Fig. 6)? He is right-handed.

The landmarks and lustre of the tympanic membranes are normal.

Case 8 is the record of a man, age 37 years, who worked for several months in an automobile plant, first in the saw mill and then in the body plant, where he did spot-welding. Both occupations, according to the patient, were very noisy, but the saw mill produced the most noise. He has never had earache or discharge from his ears. He is right-handed.

Since these occupations have not been listed in the literature as being among those which cause deafness, he was questioned further about his past occupations. Before working in this automobile plant, he had for 10 years been employed as chauffeur on the police force. While he was thus employed he spent a good deal of time in the police shooting gallery mentioned above. He said he "was a fiend for shooting," and became quite a good marksman. He did not notice at any time that this pistol shooting affected his hearing.

The absence of records of tests at intervals leaves the exact cause of this patient's deafness rather conjectural. One cannot help thinking that the target practice should be under suspicion.

The tympanic membranes are somewhat thickened, but the ossicles move well on each side.

TELEPHONE DEAFNESS—EVIDENCE FROM THE LITERATURE.

The *Journal of the American Medical Association* for July 28, 1934,[83] contains the following communication: "To the Editor:—I have a patient who claims that, following the picking up of a telephone receiver and the crackling that occurs as the operator rings the number, his ear drum burst. Otoscopic examination did show a ruptured ear drum. He is at present suing the telephone company for the accident and impaired hearing. Can you cite similar cases in medical literature? Is such an occurrence possible? (Signed) Milton Wolpert, M.D., Chester, W. Va." "Answer:—It seems rather strange that vibrations of the telephone receiver diaphragm should cause such a change in air pressure of the external auditory canal that rupture of the drum membrane would ensue. *The only reference in the literature dealing with changes*

in the ear due to the use of the telephone is: Moriez, A.: Auditory and Neurologic Disturbances Caused by Traumatizing Telephone Noises: The Nature of the Noises and the Mode of Operation. Rev. d'oto.-neuro.-ophth. II; 171. (March) 1933." (Italics by writer.)

A similar query and reply appeared in the issue of Jan. 30, 1937.[84]

It is strange indeed that such an answer should appear in response to a communication to this most excellent journal, for there are frequent references to such accidents in the otological literature, reports which must be accepted as coming from competent observers. In addition to these individual case reports, several extensive studies of the hearing of telephone operators have appeared from time to time. There is little doubt that such accidents do occur, but, with the improvement in telephone equipment, it must be said that such accidents are not as common today as they were at the turn of the century.

White,[153] in a paper before the American Otological Society, which, he said, contains "Everything which has been published on the subject," reported a case of a woman who claimed that while she "was listening to a phone was shocked, which injured her ear and head and resulted in a disturbance in her equilibrium with physical pain and general disability." When White examined this patient 19 days after the accident, he found, "No deafness, no nystagmus and no ataxia, and was unable to verify her contention, having only her word for it. I regard it as a hysterical manifestation." He concluded, "that damage to the ear could possibly result from using the telephone during an electric storm, or from electric shock by a short circuit from a high tension wire, but, judging by reports of such cases, the damage would only be temporary." Unfortunately for the present study, and perhaps for his patient, White failed to record the results of any hearing tests. The fact that he could find "no deafness" leaves the present investigator just a little undecided as to the inclusiveness of his tests.

Jack,[80] in discussing White's paper, reported that he had seen three cases, all coming from using the telephone for long distance calls. They put the receiver to their ears and "in the

process received a blow (some describe it as a blow and some as a shock) which knocked them over. They were dizzy. When they regained their composure, to a certain degree, they found that they had marked tinnitus in their ear, and shortly after that their hearing was materially impaired . . . the deafness is what I call permanent . . . it is hard for me to believe that they are manifestations of anything neurotic."

Politzer,[115] with his accustomed thoroughness, mentioned the use of the telephone as a possible cause of deafness. He stated, "The disturbances of hearing produced by the continuous use of the telephone must be reckoned among the anomalies of hearing brought about by the action of sound. . . . It is probable that the disturbances of hearing and nervous symptoms are induced by the intense cracking noise of the switchboard, which is often experienced in making the connection." Politzer found that in 26 per cent of 371 telephone operators with normal hearing "there was a retraction of the membrana tympani in the ear used in telephoning." He referred to a study made by Blake, who reported to the British Society of Telephone Engineers in London in 1878. This report was not available for this study, but, in 1888, Blake[17] stated, "The habitual use of the telephone would be prejudicial to the hearing in many cases where the hearing was already impaired, was based on the well known facts of the process of accommodation in the middle-ear for the transmission of sounds of low intensity." Shambaugh and Knudson[135] saw a case of diplacusis in a woman "with defective hearing in the left ear which had resulted from a severe electric shock from a telephone receiver. . . . The shock rendered her unconscious for about one hour." In the affected ear she could hear whisper close to the ear, while in the opposite ear it could be heard at 3 meters. "The deafness had been nearly complete after the shock, but hearing had gradually returned." Robinson[122] mentioned a case of a man who, while using the telephone, received a shock of lightning through the instrument which rendered him unconscious. When examination of his hearing was possible, he had 0/36 of normal hearing. Thirteen months later, the hearing had recovered to 12/36 of normal. Pierce[111] mentioned a similar case, a woman who received a shock from a thunderclap while using the telephone. She was affected with "syncope and deafness, accompanied by a feeling of dizziness, nausea and tinnitus. These symptoms,

with the exception of the deafness, disappeared after a few minutes. After 14 days, the normal hearing was restored." He failed to cite the tests used in determining the final hearing power. Gelle[58] observed the deleterious effects on hearing from the use of the telephone and thought they were due "either to the excessive strength and closeness of the sound or to the fatigue that is caused by the attention required for this particular transmission of sound." He concluded, "The influence of the telephonic sound is analagous to the well known observations of machinists. . . . The conducting and nervous apparatuses are equally affected, as well as the general nervous system." Capart,[31] in 1911, stated, "All agree that the danger is gradually diminishing with the perfection of the instrument. However, there are some accidents which are unavoidable even under normal conditions, such as magnetic currents, when contact is produced, which pass into the receiver and produce a very loud metallic sound." Braunstein,[22] in 1904, after examining the ears of 160 employees and officials of the Munich Telephone Co., stated, "The use of the telephone does not exert an unfavorable influence on the healthy ear," but conceded that "severe injuries to one telephoning might result from electric discharge during showers." He said also, "These injuries should not produce permanent changes if properly cared for." Blegvad[19] thought that sudden loud noises while the telephone was being used might aggravate or produce ear disease, but stated that it is not proven that the regular use of the telephone exerted an unfavorable influence. He examined the ears of 371 telephone girls and could not discover any reduction in hearing which resulted from the use of the telephone in their employment. He reported that he had seen no case of serious injury from the effects of lightning striking the telephone line. Kober and Hanson[89] state, "A more important group of injuries is caused by sudden and violent acoustic effects upon the auditory nerve of a person using the receiver. This phenomena may be caused by the entrance of a high tension current into the telephone receiver, by breaking of a power or light cable, or during an electric storm." Koelsch[91] states that in Germany during 1907 there were eignt telephone operators injured through the effects of high-tension currents, seven by other induced currents, and two others from the effects of lightning striking the telephone line." (Quoted from Kober and Hanson.) Bezold

and Siebenmann[14] make this statement, "Electric traumatisms to the acoustic nerve without any noticeable injury occur sometimes from strokes of lightning, or through the telephone during a thunderstorm." Harris,[71] in 1897, mentions a case of internal ear deafness resulting from the use of the telephone. Koetter, [92] in 1910, had for a patient a stenographer whose tympanic membrane showed a tear in the posterior-interior quadrant, resulting from a loud popping sound while she was using the telephone. The hearing for whisper which had been diminished by the accident returned to normal in six weeks. Westmacott[151] likens the deafness from telephone noises to that found in boilermakers. Bernhart[10] conceded that deafness might occur from the use of the telephone but he had seen no cases of this type nor had he been able to locate any in the literature. Cott,[39] in 1917, reported three cases of deafness from accidents occurring while the telephone was being used. In the first there was no perforation of the drum membrane, but deafness was present 16 months after the accident. The second case was deaf in both ears after a similar accident. The hysterical deafness in the unaffected side soon returned to normal, but the deafness in the affected side was permanent. The third case could, after four months, use the telephone in conversation, but the hearing was diminished.

TELEPHONE DEAFNESS—AUDIOMETRIC STUDIES.

Case 1 is the record of a woman, age 46 years, a supervisor in a telephone office. She has been in this employment for over 20 years and has been promoted to her present rank from that of operator. She has noticed some difficulty in hearing for more than two years. In her work, she has used the telephone at both ears. At the present time, she uses the right in order to leave the left open so that she may hear when the girls in the office speak to her. Her chief difficulty as operator is in distinguishing between 3's and 2's. She has no idea as to the probable cause of her deafness, unless it be the pressure of the receiver at her ears. Ten years ago, lightning struck the cable leading to the switchboard at which she was working. The intense noise of the receiver made her ear ache for a couple of days, but she thought little of it and did not report the accident to her supervisor. She states that many times she has received some "terrible whacks" in her ears, but she remembers no specific instance. There was no question of compensation in this case.

The drum membranes are normal.

Case 2 is the record of a telephone operator, age 28 years. When she entered this occupation ten years ago, she had, with the permission of her superior, used the telephone at her right ear because she was somewhat hard-of-hearing in the left, the result of otitis media in childhood. Two and one-half years ago, she noted some difficulty in

hearing in the right ear and her supervisor began to receive complaints that she was giving the wrong numbers. She was referred to the telephone company physician, who placed her on furlough and referred her for audiometric tests. There was no thought in her mind as to compensation for injury associated with her occupation. The depression was on, and her desire was to hold her job, for she was one of the principal wage earners in her family. When this record was secured, because of the contour of the curve for the right ear, trauma was suspected. As discreetly as possible, she was questioned, and gave the following additional history. Two and one-half years ago, she was called one

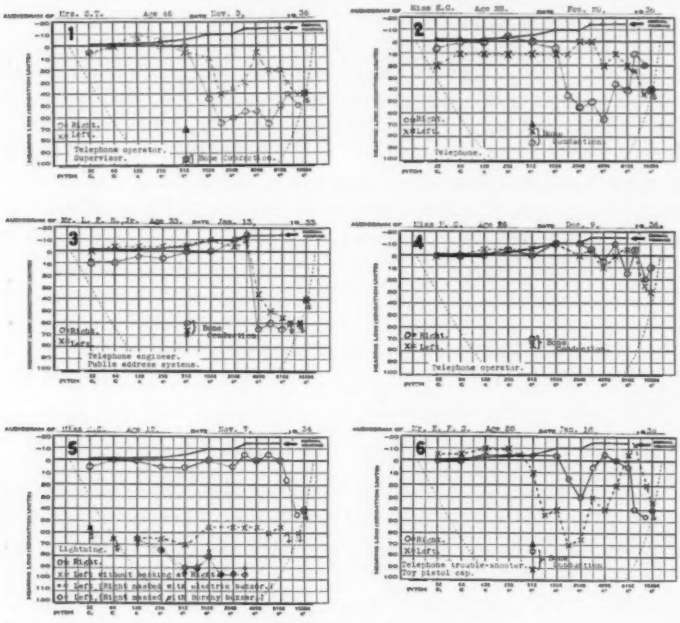

Fig. 6. Records of deafness following accidents while using the telephone.

evening by the wire chief to assist in checking the trunk lines. While she was so doing, the noises which came through the receiver she described as being "terrible," due to the methods used by the wire chief. Her ear rang during the rest of the night while she was on duty. The experience was so painful that afterward when she saw the wire chief about to call for assistance, she made herself busy about other duties so that she would not be called. It was shortly after this experience that complaints began to come in about her work.

Three weeks after this test was made, a second test showed no change in her hearing. The furlough was apparently not helping in the restoration of her hearing. She became apprehensive and feared that

she would lose her position. It was suggested to her that she ask permission of her supervisor to use a double head-set. Her chief difficulty, according to the hearing curve, was the inability to hear the high tones of the voice range in the right ear. These tones, while not quite normal in the left, are heard much better than in the right. It was thought if she used a double head-set, she would hear in her left ear the tones which she was missing in the right and thus be able to carry on. She returned for a third test ten weeks later. The hearing had not changed, but with the double head-set she was able to do her work with complete satisfaction.

Case 3: This record is that of a man, age 33 years, who was referred for tests some four years ago. The peculiar contour of the curves caused considerable comment and speculation as to the probable cause at that time. Trauma was not suspected and no attempt was made to secure any such history. So many similar curves have been secured since that time, all with a definite history of trauma; in reviewing the records, this was again brought to light, and the history reviewed. He is a telephone engineer, and up to a short time before this test was made he had considered his hearing normal. While working, he discovered that his fellow employees could hear high tones which were inaudible to him. His chief work is the installation of public address systems. Other than this, we have no record of his occupation. It is suspected now that these curves came as a result of trauma, but it has not been possible to ascertain if the deafness has progressed, or to question him more thoroughly. This record is included in the group to illustrate the fact that histories are often inadequate to bring out the pertinent facts, and it may not be entirely due to the patient's answers.

The drum membranes were normal.

Case 4: This record was included in the group because of a limited attempt to determine if all telephone operators showed similar hearing defects. This young woman, age 26 years, has been a switchboard operator for nine years. Needless to say, there has been no complaint that she gives the wrong numbers to her patrons. The line leading to her switchboard comes from an underground cable. She says that she has often had people ring in her ears, and sounds come through the receiver which are quite loud, but she has noted no hearing loss as a consequence. The high tone loss here is small and quite unlike the others of the group. Three other operators who have been employed for a shorter time have excellent hearing. It cannot be said the continual use of the telephone under ordinary conditions causes deafness, at least not in the time that these young ladies have been in that employment.

Case 5: This is the record of a patient in a medico-legal telephone case. Malingering was suspected, even though she was but 12 years of age. Two months prior to the date of these tests, the patient was using the telephone at her home in a small country town. While she was holding the receiver at her ear, lightning struck the telephone line. The telephone was torn from the wall. The child, according to her mother, was knocked across the room. Her face, head and hair were burned, her nose was fractured in the fall, and she was unconscious. She was taken to a hospital, where she regained consciousness the following morning. She was dizzy, and vomited at times, during the following week.

The problems in testing this patient were to eliminate the possibility of malingering and to determine the exact status of the hearing in the injured ear. The unaffected right ear was tested first and gave the curve of circles. Next, the telephone was placed at the left ear and the curve of crosses secured. She was quite consistent and, when checked,

gave almost identical results. This in itself was almost sufficient to prove she was not malingering, for malingerers of unilateral deafness often claim they hear nothing when the deafened ear is being tested. If they do indicate some hearing, they will not give two identical thresholds for the same tone on repeated tests, for they cannot remember tonal intensities. The curve for the left ear was approximately 60 sensation units lower than that for the right, and, with this great difference, it was suspected that this was a "shadow curve," i.e., it represented the amount of hearing around and through her head in the unaffected ear. The good ear was then masked with a small electric buzzer, ordinarily used for this purpose, and the poorer ear tested again, and the curve of dots obtained. Next, the good ear was masked with a loud Barany buzzer and the test repeated. The results of this third test are indicated by the squares on the chart. It can be seen that the last two tests, one with the good ear masked with the electric buzzer and the other with the Barany buzzer for masking, agree almost exactly. It was evident that the patient was not malingering and that the curve of dots and squares represents closely the actual hearing in the poorer ear. She could not, by memory alone, have reproduced these curves so closely when the opposite ear was masked with two such widely different types of noise. In addition to this test, when she attempted to speak and the good ear was masked with the Barany buzzer, she lost control of her vocal output. This is also a fair test for malingering. She could hear loud shouts in the poor ear when the good ear was masked, but could not understand the shouted words. The Weber test was questionably referred to the poorer ear, the Rinné test was positive on the right and indeterminate on the left, for she could not be sure that she heard the fork by air-conduction when the right was masked with the Barany buzzer, but she could hear it by bone-conduction.

The right tympanic membrane was slightly retracted and slightly lustreless. There are no perforations or discharge. The left membrane is flushed and glistening, and appears to be adherent to the medial wall of the middle-ear. The short process of the malleus and promontory of the cochlea are well outlined. There were no perforations or discharge.

Case 6 is the record of a man, age 26 years, who has been employed for nine years as a "trouble shooter" in a telephone exchange. He had noted no hearing loss until it was brought to his attention by his wife about five years ago. His occupation requires that he use a telephone receiver constantly, and he is accustomed to use it at his left ear. He notices that if he holds the right ear closed he cannot hear conversation well. He thinks that the hearing in his right ear is not affected. Often quite loud sounds have come to his ear through the telephone, but he remembers no specific time when they have been sufficiently loud to be called deafening. He was asked if he had ever been subjected to excessively loud or explosive noises of any character and gave the following story. When he was about six years of age, one day after a Fourth of July celebration he found a toy pistol cap, and wanted to determine if it would explode. Not having a pistol, he placed the cap on a piece of iron and hit it with a brick which he held in his right hand. The cap exploded, and the details of the incident are still vivid in his memory. He stated that he would never again try a similar experiment.

The fact that the hearing loss is greater in the left than in the right ear favors the use of the telephone as a possible cause. On the other hand, since the loss is bilateral but less marked in the right, it can be reasoned that the same traumatic noise affected both ears, the left more severely. He is right-handed, and has never done any hunting or shooting. In the absence of records of tests before he began his work for the telephone company, proof of the actual cause is indefinite.

DEAFNESS IN ENGINEERS, ETC.— EVIDENCE FROM THE LITERATURE.

Politzer's text[115] mentions the fact that railway trainmen are frequently hard-of-hearing and credits Duchesne with having first called attention to that fact. Zwaardemaker, according to Politzer, "Deserves the credit of having given us a complete review of aural affections in engine drivers and stokers." The atmospheric hazards to which these men are subjected are doubtless responsible for aural diseases which are in no way associated with the deafness produced by the excessively loud noises from the locomotive. The frequent occurrence of these accompanying affections apparently attracted Politzer's attention more than the effects of the acoustic trauma. Bezold and Siebenmann[13] state that the sound of the whistle of a locomotive may cause deafness. Moos,[102] in 1880, reported the results of the hearing tests on five engineers and firemen. In all, the hearing was greatly diminished for Politzer's acumeter. He associated the noises of this occupation with that of blacksmithing and boilermaking, and stated, "It seems incredible that they do not appear, when we consider what is expected from year to year from the organ of hearing by the continuous crashing of the train, the repeated hissing of steam, and the thousand repetitions of the scream of the whistle." Barr,[6] in 1890, stated that the shrill blast of a locomotive whistle could cause deafness but did not report any case. Zwaardemaker[160] states, any "locomotive engineer who had perfect hearing upon entering the service as fireman will probably suffer some diminution at the end of several years." He used a whistle with a pitch of 5,500 d.v. for his hearing tests with trainmen and found, "It is certain that they do not hear as well as a person with normal ears." He recommended that the hearing of trainmen be tested frequently to safeguard the traveling public. Stein[140] tested the hearing of 44 stokers and 20 engine drivers of various lengths of service and found that in 96 per cent the tests indicated pathologic processes of which but 11.7 per cent had lesions of the sound-conducting apparatus and 48.8 per cent had lesions of the perceptive apparatus. Lichtenberg[98] stated that some degree of impairment of hearing occurs in engine drivers and stokers and that this handicap in railroading is greater than that of color-blindness. He thought that hearing tests should be conducted at regular intervals and that all who entered this occupation should be

warned that they were likely to become hard-of-hearing. For these tests he considered the use of the voice and the watch tick to be quite insufficient. Zilliacus,[159] on the other hand, stated that railroad service had no particularly injurious effects on hearing. Sachs[129] classified railway employees and found that among conductors, baggagemen, porters, etc., deafness was no more common than among those in any similar occupation, but that engineers and firemen show considerable deafness due to their exposure to temperature and weather changes as well as to the noise and vibration of the locomotive. Roger[124] tested 48 railroad men of all ages and found that they were hard-of-hearing for the medium pitched forks and that the men who had been in this occupation for a short time had no lowering of the upper tone limit as determined by tests with the monochord. Those who had been employed for a longer period showed decreased acuity for all tones. Swann[142] found that a large percentage of locomotive workers were hard-of-hearing. This loss he concluded was caused by the repeated explosive sounds which are common in this occupation. Seely[131] says, "The American Railway Association have but one provision for these examinations. Rule 1. Applicants . . . with chronic discharges from the ear are disqualified," and recommends the use of tuning forks in tests of applicants. Andrews[1] quite bluntly states, "Hearing tests interest not a very great number of railway surgeons." No audiometric studies have been reported covering this type of occupational deafness.

DEAFNESS IN ENGINEERS, ETC.—AUDIOMETRIC STUDIES.

It has been impossible to secure any considerable number of engineers for tests in preparing this study. None of them are clinical cases, *i.e.*, usually the railroads employ their own physicians and specialists, and it would be only through their courtesy that such a study might be made. Perhaps these engineers do not desire to have the results of their hearing tests made known. The three cases presented here were picked up by chance and may not be representative, but they are presented for the evidence they contain.

Case 1 is the record of a locomotive engineer, age 53 years. He has had in all 34 years of engine service, first as fireman and later as engineer. During the last few years, he has driven his locomotive on a shuttle road between two terminals, a large part of the distance being through a tunnel, the roof of which is but a few inches above the top

of the locomotive smokestack. He says the noise of the exhaust under these conditions is like the "crack of a gun." The whistle of his own locomotive and those of passing trains, the whine of the flanges of the wheels on the rails, the popping of the safety valve and the roar of the wheels on the rails are all intensified in this tunnel. He states that nine out of ten of his fellow employees have defective hearing. He expects to be called soon for a periodic physical examination and is apprehensive that he will be unable to pass the watch test for hearing.

He has been aware of a hearing defect since 1908, but has not considered it serious until recently. In 1908, while out of employment on the railroad, he worked as hoisting engineer on a bridge construction job. This work lasted but a short time, so he took employment as a "sand-hogger" in a caisson under compressed air on the same job. He remembers that while on this job, large rocks too heavy to be elevated from the caisson were blasted. When a blast was about to be fired, the

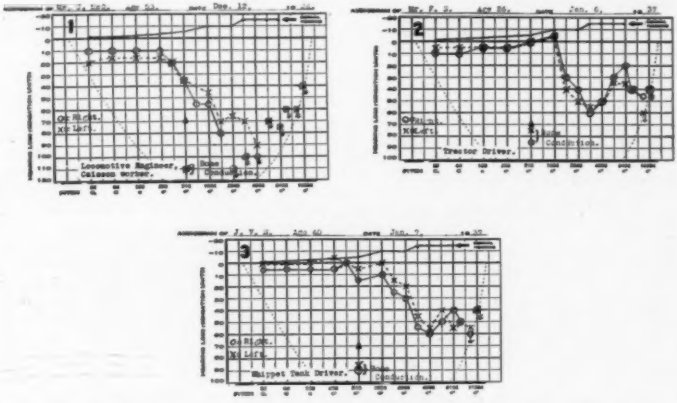

Fig. 7. Audiograms of locomotive engineers, tractor drivers, etc.

men climbed to a platform for safety, but made no attempt to protect their ears from the noise of the blast. Under these conditions, the energy of the noise is concentrated, as it were, within the walls of the caisson. He also had an attack of the "bends" while on this job. The only treatment employed at that time was to place the men in a tub of hot water for 20 minutes when they came from the compressed air chamber. He was in a hurry one day and did not remain in the bath the full 20 minutes, and the illness resulted. He dates his hearing loss from his experiences in this caisson.

This record is similar to that of others in this series who have a history of deafness from repeated acoustic insult. It is dissimilar from those where it has been assumed that the deafness resulted from formation of gas bubbles in the cochlea (see Fig. 10).

The cause of his deafness is somewhat indefinite. It apparently did not originate with his railroad service and was not sufficiently marked to prevent his re-employment after his experiences in the caisson. His work on the locomotive is not entirely responsible for his hearing loss. If it is sufficient now to cause his retirement, the railroad work must be considered responsible for the progress after the caisson experiences.

The drum membranes were somewhat dull and retracted, but both had a slight light reflex. There were no scars or calcium deposits, and the ossicles moved freely on each side.

Case 2 is the record of a farmer, age 25 years, who gave a history that his nose and ears would feel stopped up after working in the chaff and dust. He was tested as part of the routine study of allergic manifestations. When this record was secured, it was suspected that his deafness was probably of traumatic, rather than of allergic origin, and he was questioned further as to his occupation.

His work requires that he run a tractor during the plowing and threshing seasons. The tractor is old and makes "a good deal of noise." After he has worked for a day on this machine, he feels quite deaf. He has been doing this work for five years.

The trend of modern farming is to replace horses with tractors. Although those who run these tractors perhaps may not rightly be called engineers, the same hazard to hearing exists.

The drum membranes are intact, slightly thickened and moderately retracted, but otherwise normal.

Case 3 is the record of a soldier, age 40 years, who served first in the National Guard, then in the A. E. F. in France as a machine-gunner and whippet-tank driver, and finally in the forces in Nicaragua. His mother is hard-of-hearing, and he attributes his hearing loss to heredity. The curves, however, are similar to the others of this study, and traumatic origin was suspected. He did a great deal of shooting during his army service, but had this loss been due to the use of his own gun, it should have been more marked in the left ear, for he is right-handed. His experiences as a driver and machine-gunner in a whippet-tank in France are enlightening. According to his description, the driver and gunner are located in a space about two feet in diameter on the inside of a turret. The driver is seated at the motor controls, and the gunner stands immediately behind him. As soon as the motor starts, all communication between these two is by touch. They cannot hear loud shouts, because of the noise of the motor exhaust and the crash of the machinery. The muzzle of the machine gun is outside the turret. Noises sufficiently great to prevent conversation by shouting at this distance undoubtedly have a deleterious effect on hearing. Since both ears are equally affected, the evidence favors this tank service rather than that of gunfire as the probable cause of the hearing loss, and this record is included with the engineers, etc., rather than with the gunners. He states that he was in this tank during two major offensives and drove for about two full days in all.

The tympanic membranes were somewhat dull and slightly retracted.

This series of three cases is entirely inadequate for the proper consideration of this very important aspect of occupational deafness. The cases certainly are suggestive. More intensive investigation should be done. The great number of men engaged in these occupations make it vital, not only from the viewpoint of the prevention of deafness, but also from that of the protection of the public. Otological examinations with properly conducted audiometric tests can settle this problem once and for all. Much of this deafness is undoubtedly preventable. Streamlined locomotives are being built, with the enginemen placed behind protective glass and in front of the exhaust, whistle and bell, so that the sounds are carried away from them. This is a step in the right direction. If the noise of engine driving produces deafness, the enginemen should know it and be warned of that fact and be instructed how it may be prevented. In this day, when the problems of the protection and security of laborers is becoming paramount in politics, the prevention of deafness will surely soon come into prominence.

DEAFNESS IN AVIATORS—EVIDENCE FROM THE LITERATURE.

There are, as far as could be determined in this partial survey of the literature, no audiometric studies in the English language which demonstrate the effects of the noises of airplanes on hearing, but that they do adversely affect hearing is well known. Bauer,[8] in 1926, stated, "The constant roar of a high powered motor causes diminution of hearing. The pilot who has no protective device in his ears will invariably be markedly deaf. This deafness gradually wears off after a few hours. Constant flying without protection results in permanent impairment of hearing. A large proportion of the flyers in the United States Air Service show diminished hearing. This deafness can be prevented by using a suitable device. . . . A simple and very satisfactory means of protecting the ears is one described by Scott. It consists of sewing a lady's powder-puff in each ear-lap of the helmet. . . . When the helmet is firmly fastened these powder-puffs cover the ear satisfactorily and eliminate the excessive roar of the motors." And again he states, "All aviators become deaf and, if the candidate is deaf to start with, he will soon reach the stage where he cannot perform his other military functions. After a flyer is once trained, so long as he has sufficient hearing for his other military duties he may be allowed to fly. For civilian aviators, it is thought that a third of normal hearing is sufficient." This paints no pleasant picture for the ultimate outcome of the hearing of aviators. Some writers state that the deafness in aviators is not permanent. Rankin[110] stated that it is "always of temporary nature," as is also that of the mechanics who test the engines before their installment in the planes.

These two opposite statements again illustrate the disputes which have arisen between authorities who have made a study of various aspects of occupational deafness.

Scott[130] considered the deafness temporary and stated that it lasted "from one to several hours." According to this writer, the deafness is not "due to the changes in atmospheric pressure." Balla[3] "Doubts that a professional deafness exists" in aviators and thought that in those who did become deaf the chief cause was that "there existed nasopharyngeal conditions of such a nature as to be a predisposing factor for auricular disturbance." (Translation by writer.) He quotes Gradenigo as noting certain amount of deafness at the "fourth

and fifth octaves." Troena[146] in a statistical study stated, "Clinically, therefore, it seems to me that there exists a remarkable similarity between the clinical picture of cochlear deafness noted in aviators and the classical picture of professional deafness peculiar to noisy trades," and that "This special type of deafness must be considered—when other factors are excluded—as a type of deafness associated with the profession of aviation." (Translation by writer.) Blake[18] says, "In aviators the rapid alterations of altitude, productive of circulatory changes in the middle-ear and labyrinth, as evidenced by the variable subjective noises, the resultant nervous strain with accompanying secondary contraction of the tensor tympani muscle and the subjection to the continuous noise of the motor are all conditions which are prejudicial, if only tem-

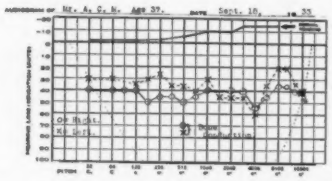

Fig. 8. Audiogram showing the hearing of an internationally famous aviator. This record shows an entirely different type of hearing loss from that illustrated in the preceding figures and a diagnosis of otosclerosis was made.

porary, to the hearing power, and when frequently repeated entail permanent results."

DEAFNESS IN AVIATORS—AUDIOMETRIC STUDIES.

In this as in other modern occupations it has not been possible to secure any large number of workers for tests. The recent reports of commissions who have investigated airplane accidents often mention the dependance of commercial aviators upon the directional beam for guidance. Is it possible that some of these pilots have been unable to hear these directional beams? In no report has there been a statement concerning this human factor as a possible cause for airplane accidents. It would seem that with the use of the directional beam as a guide in blind flying, one of the most essential requirements for commercial aviators should be good hearing.

Case 1 shows the hearing of an internationally famed pilot, age 37 years, who has been flying since 1917. The Rinné test gave a negative response in both ears and bone-conduction was very good. He reported that the otologist whom he had last consulted had told him that his deafness resulted from his flying and that it was called boilermakers' deafness. He stated that he could hear better when he was flying or in the subway. There is no history of deafness in his family. He remembers vaguely that he had earache in childhood. He has continuous head noises. The tympanic membranes show very moderate retraction, they are thin and grey, with good light reflex. There is a scarred area posterior to the malleus on the left side. The motion of the ossicles with Siegle's otoscope is good. The Eustachian tubes were clear.

The appearance of the tympanic membranes in conjunction with the history and the results of these tests caused the otologist who saw him in St. Louis to make a diagnosis of otosclerosis. The only point of similarity between the results of these tests and those of others in the group is a dip at 4,096 d.v., but this dip is so slight that perhaps it should not even be mentioned.

This description must not be interpreted to indicate the belief that the noises of the airplane cause deafness with symptoms similar to those of otosclerosis. Until more complete information is available concerning the hearing of aviators, the conclusion is forced in this case that the otosclerosis is independent of the influences of this occupation. It has been stated that adhesive processes of the middle-ear may serve to protect the inner ear from the traumatic effects of loud noises in the same manner as a plug of cotton in the external canal. It may also be possible that a bony fixation of the stapes, such as occurs in otosclerosis, may act similarly.

See also Cases 6 and 9 of Fig. 4.

MISCELLANEOUS—BLOWS ON THE EAR, ETC.

EVIDENCE FROM THE LITERATURE.

Green,[64] in 1872, reported ten cases of injury to the ear from "External Violence" and included in the group those whose injury resulted from explosions of gas bags, laboratory explosions, boxing the ears, being hit by a snowball, by a policeman's club and by a fall. In 1886, the *Lancet*[96] contained this warning, "Teachers and others would do well to remember that permanent deafness has been caused by such a homely reproof as a box on the ear." Roosa,[125] in 1887, described a case in which the deafness was ascribed to an accident which occurred while the patient was listening to a voice through a speaking tube. Coggin[35] reported a case of absolute deafness

in the left ear of a patient who had been struck on the right side of his head. Sexton,[132] in 1897, found records of 51 cases among patients of his practice who were deafened as a result of having their ears boxed. He states that usually the tympanic membranes were ruptured and attributed the deafness to the subsequent inflammatory processes. Richards[120] and Heiman[73] also reported cases of deafness following blows on the ear. Woodruff[157] had a case in whom the deafness resulted from striking the ear against a door knob during a fall. Jürgens[85] reported 13 cases of injury to the ear resulting from explosion of bombs. He found the ear on the side closer to the explosion was affected more than that on the opposite side. He found the deafness most marked for low tones and there was a tendency to improvement and recovery of the hearing after these accidents. Blake,[18] in 1916, stated, "The ruptures of the drum head incident to diving, in the open air, are commonly the results of accidents produced by the sudden condensation of air in the external canal at the moment of striking upon, or entering the water." Egeblad,[49] in 1930, reported a case of injury to the ears of a man who received an electric shock when he held a bar of iron against a 220 volt D. C. motor. Spear,[139] in 1887, reported a case of unilateral deafness in a girl resulting from being hit on the ear by a snowball. Braislin[21] had two cases of perforations of the drum membrane in men who were shocked by lightning when they were coming out of the surf from swimming. He said both made a rapid and perfect recovery. Rugani[127] says that deafness might result from a stroke of lightning or a thunder-bolt. These might also cause rupture of the tympanic membrane, disarticulation of the ossicles and labyrinthine disturbances. Packard[110] reported cases of traumatic perforations from a wave in the sea, from blows on the ear, and in a caisson worker. He has, with this article, a bibliography of 63 titles on this subject. Connal[37] reported a case of perforation of the drum membrane in a professional diver.

Shambaugh,[134] in 1908, in his description of the physiology of the cochlea recognized the possibility that the loud noise of a whistle might produce deafness. He thought the defect in hearing would be of limited extent and stated, "The disturbance in the function of hearing arising from an injury produced by a shrill whistle or an explosion near the ear are also readily explained." Again in 1936,[136] he stated, "We have

seen an abrupt island of deafness in a patient exposed without warning to a shrill locomotive whistle, while cases of occupational nerve deafness often show a marked loss for the tones of 2,048 d.v. and 4,096 d.v., with relatively good hearing below and above this island." Giron[60] found in a certain community in France that an unusually large number of men were being excused from military training because of deafness. Upon investigation, he found that in caring for their vineyards they were exposed to the noise of steam whistles on the boilers in which spraying solutions were prepared. Horne[77] says, "Even the shriek of a railway whistle, especially when passing through a covered station, may produce serious deafness." Others have also mentioned the deafness produced by a locomotive whistle. (See section on deafness in engineers, etc.)

MISCELLANEOUS—AUDIOMETRIC STUDIES.

Case 1 is the record of a groceryman, age 29 years, who wished to take up the study of music as an avocation. His music master found it difficult to teach him to sing in pitch, and thought his hearing might be defective. He gave this history: His hearing loss was first noted at the age of 15 years, when he discovered that he could not hear his watch in his right ear. Some time before this, while he was a student in a parochial school, he was struck across the right side of his face and ear by the playground supervisor for some infraction of rules. He was knocked down, was taken home, where he was ill for several days. Later he returned to school with apparently no great hearing loss. He has no tinnitus and does not remember that his ear discharged at the time. No otologist has seen this patient and no description of the drum membrane has been secured.

Case 2 is the record of a woman, age 30 years, who also was deafened by a blow on the ear. The hearing curve is different from that of Case 1, and indicates different pathology. The history is as follows: Four months prior to the date given, she was swimming with a party of friends. One of her companions, thinking to splash water on her, struck her over the right ear with his open hand, which was covered and perhaps partly filled with water. The pain was not great immediately after the accident. Within three hours, she consulted a physician, who found the drum membrane had been ruptured. Later, infection appeared and the ear has been discharging continuously since that time. The curve shows a marked loss for all tones in the range, but at 4,096 d.v. the loss is greatest. The highest tones are not heard at the maximum intensity of the audiometer. She still complains of a continuous tinnitus like the roar of a sea-shell.

Definite proof that the drum membrane was not ruptured before the accident is not available, although she denied any previous ear trouble.

The discharge had ceased on Nov. 23. The ear showed definite evidence of infection, with rather extensive destruction of the drumhead in the anterior and inferior portions. The malleus appeared to have been pushed inward and backward against the inner tympanic wall.

Case 3: This patient, a man, age 55 years, complained of loss of hearing in his right ear only. There was a history of apoplectiform

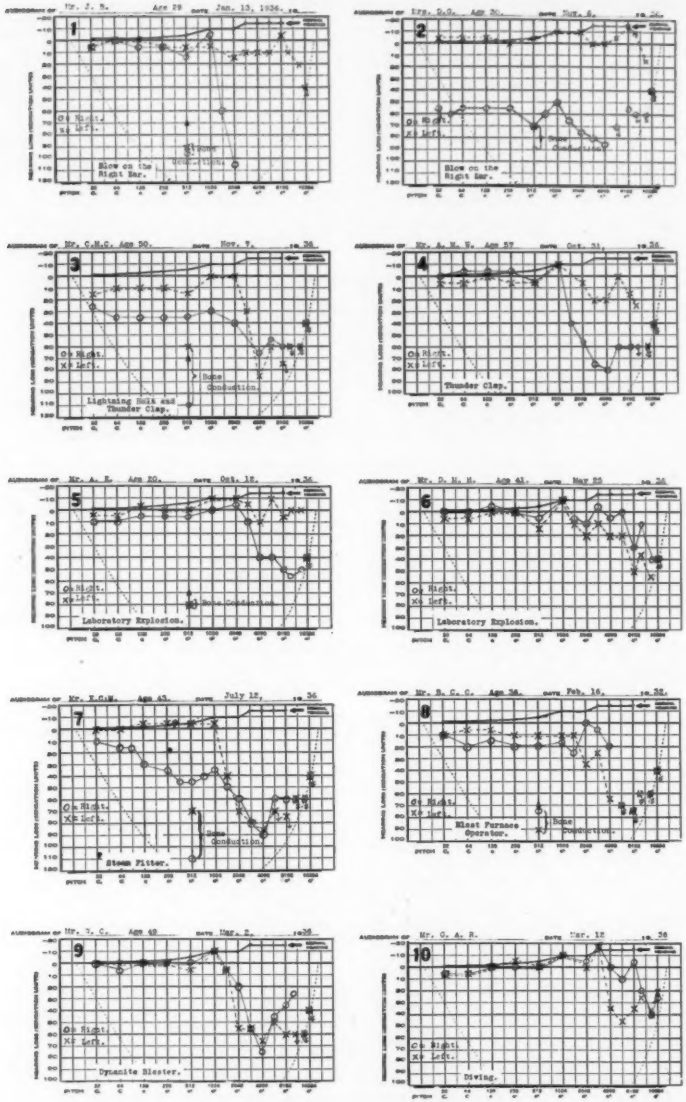

Fig. 9. Audiograms showing deafness resulting from blows on the ear, laboratory explosions, thunder-clap, etc.

seizures, and, at the time of the tests, a tumor of the cerebello-pontine angle was suspected. The curve for the right ear shows some loss for all tones, but the greatest loss is at 4,096 d.v. The left ear shows slight loss for tones below 2,048 d.v., and shows a similar drop at 4,096 d.v., with even greater loss than that in the right at this point. This sharp loss in both ears aroused suspicion that in addition to the evidence of intracranial involvement, there was a probability of acoustic trauma of some nature, and he was questioned along this line. At first, because of the prominence of the symptoms of dizziness, etc., no history of trauma could be elicited. Later, he remembered this accident. Five years ago, during a rainstorm, he was attempting to enter his cottage in the country. He had lost his house key and was forcing a window, when lightning struck the cottage. He was severely shocked. The roof and sides of the cottage were badly damaged. The only effects he noticed were the force of the electric shock and the extreme noise of the thunderclap.

The drum membranes showed only moderate retraction, with good motion of the ossicles and drums. His otologist ruled out all possibilities of his symptoms being caused by an angle tumor and concluded that they were the result of the accident.

Case 4: This man, age 57 years, noted his deafness 14 years ago. He remembers distinctly when he drove his first automobile that he had difficulty in understanding the conversation of those seated at his right in his car. A test made two years before showed that there had been no progress during that time. When he was first tested, he was not questioned as to the possibilities of this loss being caused by trauma, because the typical features of traumatic deafness were not recognized. At the second examination he was asked about his occupation, and nothing relevant was learned until he was asked about explosions. He then remembered the following accident: When he was 11 years of age, he was driving a team hitched to a wagon. His father was driving a second team in front of him. During a rainstorm, lightning struck a tree, 50 or 75 feet to the right and in front of him. The father, who was closer, was knocked down and rather severely shocked by the force of the thunderbolt. The patient remembers that his father was hard-of-hearing during his life, but does not remember whether the deafness antedated the shock by the thunderbolt. The patient recalls the accident vividly, but did not discover any hearing loss until 30 years later. This explanation of the cause of the deafness may appear naive, but the circumstances are reported as they occurred. He has been aware of the deafness for 14 years, and since its discovery was accidental, it may have been present before. Regardless of the time when the accident occurred and the date of its discovery, the loss could have resulted from the noise of the thunderbolt. It is probable that had the left ear been affected, it would have been discovered earlier through the use of the telephone. He is an intelligent man, the principal of one of the larger grade school in St. Louis.

Both tympanic membranes are retracted with slight prominence of the short process of the malleus. The light reflex in the left is shortened, but the membranes and ossicles move well.

Case 5 is the record of a high school boy, 20 years old, who was referred for hearing tests by the Neuro-Surgery department when it was discovered that he was unable to hear a watch in his right ear as well as in the left. He entered the hospital with a history of attacks of unconsciousness, which were preceded by buzzing and humming in his ears. No history of acoustic trauma was secured until after the tests were made and the contour of the curves studied. The marked drop at 4,096 d.v. in the right ear aroused suspicion, and he was questioned further. He is an amateur chemist and has had several rather severe explosions in his chemical laboratory. The most severe explosion oc-

curred when he dropped a piece of metallic magnesium into some water under a chemical hood. This explanation was conveyed to the referring department as a possible cause for his hearing loss.

The Neuro-Surgical diagnosis was idiopathic epilepsy. The blood Kahn was negative.

Case 6: This record shows the hearing of one of the members of the Medical School Faculty, a man, age 41 years. He has no difficulty in hearing conversation, and takes his part in the regular instructional course. This record was taken in the search for material in this study because it was known that he had had a rather severe explosion in his chemical laboratory, 18 years ago. He was quite deaf immediately after the accident, but his hearing improved until he thought it normal. The explosion, as he described it, made a terrific noise and did considerable damage to his laboratory equipment.

Case 7 is the record of a plumber and steam-fitter, age 43 years. Two months prior to the date of this test, he was repairing a safety valve on a steam boiler. The steam had been turned off and parts of the valve removed. A fellow workman unwittingly turned on the full head of steam through this open valve directly in front of him as he was working at the valve. He says that the noise of the escaping steam was "terrific." He was deafened in both ears at first, but within a short time he thought the hearing in the left ear returned to normal. He claimed damages because of the deafness in the right ear. He was not burned. Tinnitus has been continuous since the accident. Tests repeated four months later showed no change in his hearing.

It seems impossible that the noise described could produce deafness in one ear alone. There is no knowledge of the condition of his hearing before the accident. His contention that it was good is no proof, for he now claims the hearing in his left ear is good, but it is very poor for high tones.

The left tympanic membrane was thickened and moderately retracted and showed no signs of recent inflammation. The right membrane showed some congestion along the handle of the malleus and in the periphery, suggesting catarrhal process in the middle ear. It was also somewhat retracted.

Case 8 is the record of a man, age 36 years, who finds it difficult to understand conversation when several persons are speaking at the same time. He can hear his watch better in his right ear than in the left. He has been employed for 10 years in a room near a very noisy blast furnace. In 1917-18, he was often on the rifle range and did a great deal of target shooting. It is difficult to determine what part each of these occupations played in the production of his hearing loss. The fact that it is greater in the left ear favors the target practice as the principal cause since he is right-handed, but it is possible that his left ear received greater trauma from the blast furnace, due to its location.

Both drum membranes are slightly dull and just perceptibly retracted. The color is normal, and there are no scars or perforations.

Case 9 is the record of a man, age 49 years, who was brought to the hospital from a government camp with a diagnosis of influenza, but this was later proven to be malaria. He stated that for many years he had lived in a locality where malaria was often prevalent and that he had taken a great deal of quinine during malarial attacks. Because of the contour of the curves and its similarity to the others of this group, suspicion was aroused that the deafness resulted from trauma, rather than from the use of quinine. He denied all loss of hearing and thought he could hear as well as any one of his age. He complained of tinnitus, and attributed it to the use of quinine. On further questioning, he admitted that he had done a great deal of hunting since boyhood, but

he had not noticed that shooting affected his hearing in any way. Four years ago, he worked in a stone quarry. His job was to fire the charges of dynamite in blasting. Sometimes eight or ten charges would be prepared, and he would light the fuses and run for protection. After firing these blasts, his hearing would be dulled, but would apparently clear up over night.

Until more evidence is available as to the influence of quinine on hearing, the evidence favors acoustic trauma as the probable cause of his hearing loss. The tympanic membranes were somewhat dull, but otherwise normal.

Case 10 is the record of one of the internes in the department, age 31 years. He discovered this hearing loss when he was testing his own ears with the audiometer. The record is similar to others of this group, so he tried to recall any possible acoustic trauma. He has done no shooting, nor has he worked in a noisy place. No one has boxed or shouted in his ears. While diving several years ago, he lost his balance in mid-air and struck the water heavily on his left side. The force of the blow deafened him at the time, but his hearing apparently returned to normal, and the accident had been forgotten.

CAISSON DEAFNESS—EVIDENCE FROM THE LITERATURE.

Deafness as one manifestation of "caisson disease" or "bends" is not infrequent and is mentioned in some otological texts. Turner[147] states, "Caisson workers are liable to nerve deafness if compression or decompression is carried out too rapidly. The symptoms manifest themselves after the patient has left the caisson; the onset is acute, the deafness being accompanied by giddiness, tinnitus and vomiting. The deafness is rarely permanent. The pathological changes are due either to a hemorrhage into the labyrinth or to the development of emboli in that organ. Airmen, mountaineers and divers are liable to similar lesions." The statements that these men are liable to nerve deafness and that it is rarely permanent are somewhat contradictory. Hill[74] in his text on "Caisson Sickness" mentions a case of apoplectiform deafness. He also gives tables from the work of Heller, Magen and von Schrötter, who studied the men during the construction of a bridge at Nusdorf. According to these tables, ear manifestations occurred in 21.5 per cent of the workers. Bassoe[7] states that 87 of 161 men who were affected with caisson sickness gave a history of ear affections and 65 of these had more or less impairment of hearing. "Eight with more or less marked permanent deafness were referred to Dr. G. W. Boot for otological examination. In every one a diagnosis of labyrinthine deafness was arrived at." Erdman[51] gave us what is generally accepted by physiologists today as the probable cause of deafness in these cases. He says, "Deafness and vertigo may follow the formation of bubbles in the labyrinth

or hemorrhage in the same situation." His article contains an excellent review of this subject and has a bibliography of 18 titles. Boot[20] says he examined 13 of these cases and concluded, "The most characteristic result of working under compressed air is a loss of a considerable portion of the upper range of hearing, with marked loss of bone-conduction. In a few cases the whole organ of Corti was apparently destroyed after coming out of the air-lock. As a rule, the loss is gradual, and is comparable to that in boilermakers' deafness." Kober and Hanson's text[90] has a chapter on "Compressed Air Illness" written by Erdman. In this he states, "I know of but three cases (in 3693) of ruptured membrani tympani and with persistent impairment in hearing; one of these developed an obstinate otitis media purulenta. . . . However, cases of labyrinthine deafness are reported as setting in after decompression, and due to bubbles in the labyrinth; such cases of which I saw no permanent ones, would be properly classed as symptoms of this disease." Vail[149] saw two cases of traumatic rupture of the tympanic membranes in caisson workers and gave the audiograms showing the amount of the defect in hearing. He says, "There are two main types of ear trauma, one where the symptoms are part of the symptom complex of caisson disease and produced by nitogren bubbles in the cochlea and vestibular apparatus, the other where they are directly due to the action of the compressed air."

Politzer[113] states, "Excessive variations in the air pressure, which occur in caisson workers, divers, and aeronauts, must be classed among the influences which are injurious to the organ of hearing. As a result of the excessive increase or decrease in the air-pressure, lacerations of the membrana tympani, as well as extravasations of blood into the tympanum and labyrinth, have been known to occur." And again,[114] "The majority of aural affections in caisson workers occur, according to the investigations of Herm. von Schrotter, Heller and Mager, during the stage of decompression, if this is carried out too rapidly. In addition to the lesions already mentioned, gas emboli are induced during the stage of decompression in consequence of the development of gas in the blood. These gas emboli may cause permanent disturbances of hearing through ischemia, and through local destruction of the central fibres of the auditory tract, the auditory nerve and its branches."

CAISSON DEAFNESS—AUDIOMETRIC STUDIES.

Case 1 shows the record in a case of caisson deafness. Five weeks before this test was made, this patient, age 35 years, was employed in a caisson under pressures, according to his statement, up to 35 pounds. Due to a sudden accidental lowering of the pressure, the right tympanic membrane was ruptured. Dizziness was not at first a prominent symptom. His mental processes appear to be slowed. He was placed in a hospital for observation for ten days and the hearing again tested, and results similar to those of the first test were obtained. Bone-conduction was markedly diminished, and the Rinné test gave the positive result on each side. If the pathology indicated by these curves resulted from the formation of gas bubbles within the cochlear structures, they apparently caused almost equal damage throughout the entire length of the cochlea, for the hearing loss is quite uniform throughout the entire range of the audiometer.

The right drum membrane was slightly red and thickened in Shrapnell's area, but otherwise normal. The left was slightly retracted.

When temporal bones are being prepared for microscopic study, it is difficult to prevent the formation of bubbles in the spaces of the inner

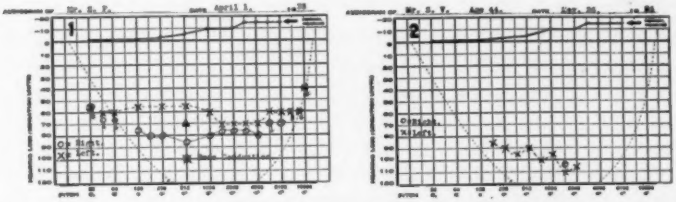

Fig. 10. Audiograms showing deafness in caisson workers. These curves have a different contour than those from patients in whom the deafness is assumed to have been caused by extremely loud noises.

ear. Such bubbles, when they form, often change the contour of the structures of the inner ear. The delicate membranes may be stretched or torn and pushed out of their normal location. It seems probable that the formation of gas bubbles in the ear of the living might be equally destructive, with serious consequences to the function of the hearing mechanism.

Case 2: This is the record of a man, age 44 years. Seven months before this test was made, he was engaged in sewer construction, where compressed air of varying pressures was used to prevent water from entering the ditch. Unwittingly, the boy in charge of the air-lock opened it to permit the entrance of construction materials. With the sudden change of air pressures, this man fell to the ground, pressing his hands against his ears because of the extreme pain. He still is dizzy at times and has intense pains in the back of his head. His mental processes and movements seem slower than normal. He speaks loudly, as if he did not hear his own voice well. He claims that he has never before been ill. The audiogram shows just one tone audible in the right ear. There is a larger island of hearing in the left, extending over three and one-half octaves. In the right, he could not hear loud shouts close to his ear. With the left, he could hear and repeat shouted words, but could not hear the loudest whisper.

The ear drums are somewhat thickened, as if there had been some previous otitis media, although this is denied. In the right, there is evidence of an old, healed perforation. On several occasions since the accident, even recently, there have appeared small hemorrhages in the drum membranes from the rupture of small vessels which have been previously weakened.

COMMENTS ON THE EFFECTS OF AGE.

The loss of hearing which is often an accompaniment of advanced age has been so universally recognized that it has been given the name presbycusis. Just what part acoustic trauma plays in the production of this deafness remains to be determined. It is well known that everyone in modern life is at times subjected to loud noises. Is the cumulative effects of these noises, any one of which may not in itself cause a measurable hearing defect, sufficient to result eventually in a certain degree of deafness? Or is there something inherent in the hearing organ which will eventually result in decreased acuity of hearing? It is inconceivable that the typical hearing loss which is shown in the accompanying audiograms should be attributed to the effects of advancing age. Only three of the cases presented are over 60 years of age and the youngest showing a loss for high tones without a corresponding loss for low is a pre-adolescent youth, age 13 years. Concerning this phenomenon, Wolf wrote in 1874,[156] "It is conceivable that such disturbances of innervation should make their appearance at this time of life, in which men are exposed to the various harmful influences of their trade or of their mode of life, such as the continued hearing of loud tones (in the case of musicians), or of loud noises, which act powerfully on the auditory nerves (as in the case of locomotive drivers, tin-smiths, copper-smiths, boilermakers, or telegraph operators); or such influences, commencing in youth, may cause profound nervous alterations in middle life. . . . The fact that the higher series of tones was more frequently visited by these affections than the middle and lower series is perhaps explicable from the fact already mentioned, that an abnormal increase in the labyrinthine pressure . . . which . . . may . . . be assumed to exist interferes with the vibrations of the fenestra chiefly for the higher tones of the scale." Bezold,[15] in 1894, wrote as follows, "With the old people, there is a marked difference between the sexes. The number of men who perceive whispering at 1 m. to $\frac{1}{\infty}$ is relatively much larger than the number of women. The explanation is to be sought in the many hurtful

influences of their calling, noisy occupations, detonations, trauma, alcohol, nicotine, etc. . . . In old age, bone-conduction in itself does not experience a reduction, but sinks proportionately with the lessening of hearing distance." Bruhl's text[23] also mentions the loss of acuity for high tones so frequently found in elderly people. Bezold and Siebenmann's text,[11] written in 1907, states as follows, "A certain decrease of the power of hearing is a regular senile phenomenon (presbycusis). It is, however, not strictly peculiar to senility, but may occur earlier, corresponding to a general law that senile degeneration of any organ may take place in different individuals at different decades of life. Hereditary influences are evident in this regard. The beginning and the development of senile deafness frequently occur slowly and imperceptibly. Other disorders besides senile degeneration must be taken for granted when there is ringing and dizziness present. . . . Senile deafness always concerns both ears. The functional tests show a clear picture of nervous deafness, modified to such an extent that the hearing in the region of the Galton's whistle is usually considerably reduced. . . . That old people are deaf to the chirping of crickets is generally known. A defect of hearing at the lower end of the scale is rarely observed." Grant[62] states that senile changes in the ear produce deafness of the nerve type. Bunch,[30] in 1929, tested 353 hospital patients from 20 to 60 years of age, using the Western Electric Audiometer. Both males and females were represented in this group. The results indicated that in each successive decade there was an increasing loss for high tones, while the low tones were heard equally well by all groups. Rideout,[121] in 1930, made a similar study of the hearing of 221 officers in the U. S. Navy. His results are similar to those of Bunch (above). The title of his article, "Gunfire Deafness in the Navy," indicates that he attributed the loss for high tones in each decade to the traumatic effects of the noise of gunfire. Bunch,[29] in 1931, again reported a study of 468 hospital patients and could find no generalized clinical disease responsible for the increasing loss in high tones shown in each decade. Again in 1931, Bunch and Raiford[28] tested the hearing of 693 hospital patients and tabulated their results according to age, race (white and black), and sex. This study agreed essentially with the two earlier studies by Bunch except that it showed a greater loss for high tones (from 4,096 to 8,192)

in white males than in white females or in colored males or females. This appears to confirm the statement of Bezold (see above). Ciocco,[34] in 1932, confirmed these studies, using the records of tests on 1,980 hospital patients. As to the probable cause of this greater loss in white males, Ciocco says, "It may be a true sexual difference. On the other hand, although we have attempted to exclude all individuals whose occupational history was such as to give reason to suspect its influence on hearing, still it must be remembered that the male patients who comprised our material are for most part laborers whose several occupations may have exposed them in many instances to injurious noises; correspondingly, the females are, in general, housewives and have led a more sheltered life."

ANIMAL EXPERIMENTATION.

The exact function of the various parts of the organ of hearing has long been of intense interest to otologists, physiologists, psychologists, and scientists from allied fields. The so-called Helmholtz theory of hearing or some modification of it appears to have been quite generally accepted up to about 1898, when Rutherford[128] presented his "telephone" theory. The fact being quite well established that deafness could be caused in human beings by excessively loud sounds led many investigators, during the decades just before and after 1910, to resort to animal experimentation for verification of this clinical observation. Since these studies are outside the field of human hearing, they can be mentioned but briefly here. Kemp,[86] in 1935, published a critical review covering quite thoroughly the studies relating to the production of "stimulation deafness," as he called it, in animals. He limits the review to those studies in which the stimulii were pure tones. Since this review did not appear in the otological literature and may not be readily accessible to otologists, for convenience his tables are reproduced here.

In addition to preparing these tables, he gave a bibliography of 44 titles relating to this subject. In his summary, he stated, "The foregoing facts may be summarized as follows:

"*1.* People who work in extremely noisy environments are often found to be hard-of-hearing, particularly for high frequencies. One investigator has reported deafness for certain low frequencies in people who were working in an environ-

TABLE I.
A SUMMARY OF EXPERIMENTS ON GUINEA PIGS.

Name	Conditions of Exposure		Duration	Anatomical Lesion
	Tone	Intensity, Etc.*		
	Bell	(By air-cond.)	5-60 days.	No effect.
	Bell	(By bone-cond.)	5-16 days.	Atrophy organ of Corti and VIIIth nerve.
	Bell	(By bone-cond.)	3-240 days x 12 hours.	
Wittmaack 1907	Whistle	"voller Lungenkraft."	3-200 days 4 times per day.	Basal degeneration of nerve and organ of Corti.
	Whistle	"voller Lungenkraft."	Once only.	Basal degeneration of nerve and organ of Corti.
	c_3	"voller Lungenkraft."	Once daily x 28 days.	Degeneration of nerve and organ of Corti, second turn.
	c_5	5 mm. Hg. pressure.	30-120 days x 12 hours.	Ext. and int. hair cells of middle basal turn.
Yoshii 1909	b_2	4-5 mm. Hg.	30 days x 12 hours.	Same in lower second turn.
	G	4-5 mm. Hg.	30-40 days x 12 hours.	Same in upper second turn.
	Siren, c_3 to f_4	4-5 mm. Hg.	Once daily x 8—130 days.	Same in lower and upper second turns.
	Trill, f_4	4-5 mm. Hg.		Same in basal down to end.
	a_4		28 days x ? hours.	Hair cells in upper basal turn.
Marx 1909	a_3		28 days x ? hours.	Hair cells in lower basal turn.
	a_2		28 days x ? hours.	No effect.
	e_2		28 days x ? hours.	No effect.
	G		42 days x 12 hours.	Degeneration.
von Eicken	G	(Anvil removed.)	42 days x 12 hours.	No effect.
	C		42 days x 12 hours.	No effect.
	c_5	5 mm. Hg.	10 days x 10-12 hours.	Ext. hair cells upper basal turn.
	g_4	5 mm. Hg.	32 days x 10-12 hours.	Ext. hair cells upper basal and lower second.
Hössli 1911-12	c_4 and c_5	5 mm. Hg.	20 days x 10-12 hours.	Ext. hair cells lower basal and middle second.
	b_2 followed by c_5	5 mm. Hg.	21 (+ 14) days x 10-12 hours.	Ext. hair cells two lower turns.
	c_2 and c_5	5 mm. Hg.		Proximal ext. hair cells upper basal turn.
	c			No effect.
	c_0	(All directly in meatus.)		Ext. hair cell highest turn.
	c_1			Organ of Corti third turn.
Röhr 1921	c_2			Organ of Corti third turn.
	c_3			No effect.
	c_4			No effect.
	c_5			No effect.
	c_6			No effect.
Kimura 1924	c_3	Blown by carbonic acid bomb.	2-6 weeks (2 min. daily.)	Organ of Corti between two lowest turns.

*This column contains information concerning exposure conditions which might affect intensity.

TABLE II.
EXPERIMENTS ON ANIMALS OTHER THAN GUINEA PIGS.

Name	Animal	Condition of Exposure			Locus of Anatomical Lesion
		Pitch	Intensity, Etc.	Duration	
Marx 1909	Mice	Like guinea pigs except for wire.			No effects.
Grunberg 1912	Pigeons	a_3	"voller Lungenkraft."	7 days.	Second quarter.
		a_2		14 days.	Second quarter.
		a_4		7 days.	Second quarter.
		a_4		14 days.	No effects.
Hössli 1912	Apes	c_5	5 mm. Hg.	10 days x 10-12 hours.	Ext. hair cells upper basal turn.
		c_5	5 mm. Hg.	10 days x 10-12 hours.	No effects.
		c_5 and c_6	5 mm. Hg.	32 days x 10-12 hours.	No effects.
	Cats	c_5	5 mm. Hg.	19 days x 10-12 hours.	Ext. hair cells lower second turn.
		c_5	5 mm. Hg.	19 days x 10-12 hours.	No effects.
	Dogs	d_1 and d_4	5 mm. Hg.	42 days x 10-12 hours.	Upper first and lower second turn.
		c_4 followed by e_1	5 mm. Hg.	21 days x 10-12 hours. 33 days x 10-12 hours.	No effects.
Satch 1917	Pigeons	c_5	5 mm. Hg.	10-50 days x 12 hours.	No effects.
	Starlings	c_5	5 mm. Hg.	10-50 days x 12 hours.	No effects.
	and Parrots	c_1	5 mm. Hg.	10-50 days x 12 hours.	No effects.
Röhr 1921	Mice	As with guinea pigs.			No effects.
Wada 1924	Pigeons	With tensor tympani normal.		60-71 hours.	No effects.
		With tensor tympani cut.		61-63 hours.	No effects.
Kimura 1924	Mice	c_2	H_2CO_3 bomb.	2 min. daily 2-6 wks.	Organ of Corti degeneration between two lowest turns.
	Pigeons	c_3		2 min. daily 2-3 wks.	Degeneration part of papilla basilaris.

ment where, it is said, these low tones were predominant. (In such cases, the relationship between age and hearing defect needs to be considered.)

"*2.* Guinea pigs, exposed for a considerable time to tones of high intensity, have frequently been found to possess cochlear lesions. Such lesions have been produced in several instances where the stimuli have been tones of high frequency, but it is not certain that low tones are similarly effective.

"*3.* Results with exposure of other animals are not conclusive; but there is evidence that sufficiently intense and prolonged stimuli will produce cochlear lesions in white mice and pigeons, and possibly in cats, dogs, and apes."

Richardson, in 1918,[119] wrote, "After reading the reports of various experiments and findings of Wittmaack, Von Eicken, Hössli, and others, we are fairly convinced that laboratory findings cannot completely fit in with certain clinical evidences as found in connection with cases subjected to sudden and intense changes in atmospheric pressure."

So many of these studies appeared and with such varying results that Gray,[63] in 1921, wrote with perhaps a tinge of sarcasm: "Reviewing in general the whole subject, it must be admitted that though much time and thought have been devoted to it during the last 20 or 30 years, we do not appear to be any nearer a conclusion. So much is this the case that one sometimes wonders if the mental energy spent upon the problem would not be better spent in some more fruitful field of research. The study of the subject, however, offers the opportunity for excellent mental gymnastics, and further brings us into touch with spheres of intellectual activity outside our own. The most pressing need at present in the study of theories of hearing is a definite pronouncement from physiologists as to what the nerve fibre and nerve cells are capable of in regard to the conductivity of impulses, both in regard to their frequency and their quality."

Almost as if in response to this request of Gray's for more information concerning the functional activity of the nerve, Wever and Bray[152] devised their very accurate technique for demonstrating the activity of the cochlear nerve or cochlea, whichever it may be. It is not within the scope of this review to present all the ramifications through which investigators

using this technique would lead us. It is sufficient to mention but a few to illustrate again the morass of conflicting evidence which is rapidly being accumulated. Davis, *et al.*,[43] in 1935, submitted cats and guinea pigs to a tone of 600 d.v. at 85 dcb. above the human threshold for periods of time up to two months. The animals were later tested by means of the Wever-Bray method and the ears were finally examined histologically and "none of them showed significant deviations from normal or recognizable histological lesions." A second group of animals was submitted to a tone of 800 cycles at 95 dcb. for periods up to 74 days and these also were found to be normal on examination. Other groups were used experimentally in a like manner and these writers conclude somewhat indefinitely, "The results indicate that the frequency as well as the intensity of the exposure tone may be an important factor in determining whether or not the inner ear is damaged. Considerable individual differences in susceptibility are also indicated." Horton,[78] using the conditioned reflex method for tests, determined the auditory acuity of guinea pigs. Later the animals were submitted to a tone of 1,000 cycles for 110 hours. Subsequent tests showed that this intense stimulation caused a loss of sensitivity for the eight octaves (64, 128, 256, 512, 1,024, 2,048, 4,096, and 8,192 d.v.), the loss being quite uniform for all tones. Culler and Finch,[41] also using the conditioned reflex method found the following: "*1.* Protracted stimulation of 1,000 cycles is followed by a severe horizontal subsidence of auditory acuity throughout an extensive section of the audible range. (At least from 200 to 5,000 cycles.) *2.* A given quantity of sound energy extending over ten hours at 4,000 cycles brought a loss of 55 dcb. at 125 cycles, increasing from that point to a maximal loss of 104 dcb. at 4,000 cycles. *3.* The same quantity of sound energy at a rate of 250 cycles gave on the contrary no loss at any frequency from 125 to 8,000 cycles. . . . *6.* When the tonal stimulus is interrupted about once per second, it induces far greater functional loss of hearing than does a continuous tone of the same amplitude." Using the Wever-Bray technique, Davis and his group of workers[141] took audiograms of the hearing of guinea pigs before and after producing localized lesions in the cochlea by means of a fine drill. They found that the loss in sensitivity following such operative procedure was correlated with the location of the lesion. They also reported, "An

accident provided us with significant evidence as to which of the membranes of the inner ear is tuned to resonate specifically to different tones . . . a loud tone (pitch not stated) was inadvertantly introduced into the animal's ear and an elevation of the threshold to tones in the middle range resulted. . . . Since the accidental exposure to the loud tone had spoiled the ear for our purposes, we proceeded to investigate the effect of an intense *low* tone on the ear. . . . The animal was next exposed to a tone of 400 c.p.s. at 125 db. above the threshold . . . curiously, subsequent sectioning of the cochlea for microscopic examination *showed that the exposure had been sufficient to shake the organ of Corti loose from the basilar membrane.*" (Italics by writer.) It is to be regretted that these investigators did not repeat this experiment to substantiate the results. The pitch of the accidental tone is not stated and it is not possible to determine whether this tone or some other factor was responsible for the described lesion. The writers have failed in definitely establishing a cause and effect relationship.

This study is prepared in an attempt to understand more clearly the clinical aspects of the problem of traumatic deafness. At the present time, it is difficult to see any clinical application of the results of these animal experiments. Their results have been in such great disagreement and the methods of approach have been so varied that little information of practical clinical value has been obtained. This is perhaps to be expected since practically all of them have attempted to throw some light on the functional activity of the conductive or perceptive mechanisms of the organ of hearing. It seems to the writer that little information which can apply to the treatment of deafness will be determined by these methods of animal experimentation, and the best that can be hoped for is some new light on the theories of hearing. It is true that Fromm, Nylên and Zoltermann[57] have used the Wever-Bray technique in studying the deafness of humans, but their results have been of academic rather than of practical value. Insofar as such discoveries can be applied to the actual treatment of deafness, their results will be of practical value. These are not yet apparent.

PATHOLOGICAL REPORTS.

The pathology found in the ears in cases of occupational deafness should receive some consideration here. None of

the cases presented in this study have died, so this discussion is limited to a resume of the published reports.

Lesions of the tympanic membrane and simultaneous or subsequent involvement of the middle ear sometimes result from true occupational influences. These, however, are not particularly different from tympanic lesions produced by other causes and the simple recognition of the fact is sufficient for the needs of this study.

Grove[60] described the deafness which he found in persons who had suffered from skull injury as follows: "I found a loss of hearing revealed on the audiogram which is hard to explain. This loss was indicated by a more or less isolated notch in the region of the c^5 (4,096 d.v.) tone. The hearing was fairly normal below this point. . . . The best explanation I can offer for this phenomenon is that there was a more or less isolated hemorrhage in the basal coil of the cochlea. . . . The 12 patients might easily pass for persons with normal hearing, but the examination with the audiometer always reveals a loss of hearing or a tone gap at this particular point." Many of the cases in the present study reveal tonal gaps at 4,096 d.v. similar to those described by Grove, but very few have a history of head trauma. Several of Grove's cases were laborers, one was a worker in a tunnel. As in the present study, Grove had no tests prior to the accident. It does not seem impossible that the gaps described by him may have been occupational and present before the head injury.

Moos and Steinbrügge,[103] in 1881, reported the histological examination of the ears of a case to which frequent reference is made. The patient was a man, 63 years of age, who, when examined two weeks before death, was found to be deaf for high pitched tones. The nerve fibres in the two upper turns were good, but there was marked atrophy in the lower turn. The only occupational statement is that he was a mason.

Habermann,[69] in 1891, under the title, "Hardness of Hearing in Boilermakers," described the pathology in the inner ear of a man who died at the age of 75 who had for 20 years been employed in a copper works. "He found atrophy of the nerve trunks of the entire inner ear. In the basal cochlear turn there was found in the canalis ganglionis but a few ganglion cells; and the canal was filled with mucoid connective

tissue; in place of Corti's organ there was an indistinct mass of cells. Toward the cupola of the cochlea the structures were found to be normal." The condition of this man's hearing prior to death is not stated. Bruhl,[24] in 1907, described the cochlear sections of two cases diagnosed as being neural deafness before death. One of them was a blacksmith. The pathological condition found was quite similar to that described by Habermann. Bezold and Siebenmann's text[13] of 1907 states that there had been reported at that time but one case of the histological findings in professional deafness. Bryant,[26] in 1908, found reports of the "records of 26 ears which had had a functional test, autopsy and histological examination with sufficient data to warrant their consideration in this connection." Wilson,[154] in 1917, stated, "The pathology of nerve deafness from high explosives is unknown." Guild,[68] in 1918, stated, "Modern battlefields gradually cause a 'noise deafness' in which the lesions are doubtless similar to those occurring in 'boilermakers' deafness' . . . though no one has yet reported the microscopic study of such a case of noise deafness developed in the war."

The only published report in which an attempt has been made to correlate the high tone losses shown in audiometric tests with histologic studies is that by Crowe, Guild and Polvogt.[40] This study gives in tabular form a description of the pathology found in 79 ears which had audiometric tests before death and compares them with 15 "normal" ears. They divide their cases into two groups: *1.* those showing a gradually increasing loss for tones of high pitch; and *2.* those showing practically normal hearing for low pitched tones with an abrupt drop in acuity, usually near 2,048 d.v., with gradually diminished acuity for tones above this pitch. Three audiograms from cases in their study were kindly sent by Dr. Guild to the writer and are shown in Fig. 11.

Concerning the pathology in cases of "abrupt" high tone loss, these writers make these observations:

"Total atrophy of the nerve is extensive in some cases, in others it is localized. There is always total atrophy of the organ of Corti in the regions in which the nerve has entirely disappeared. . . . The transition from total absence of nerve fibres to normal nerve within a space of 1 mm. or less is often seen in this group in which there is an 'abrupt' and

marked functional loss of hearing for tones an octave apart. . . . The most distinctive histological lesion in this group of ears with an abrupt high tone loss is atrophy of the organ of Corti at some level above the last 2 mm. of the basal turn. The atrophy of the organ of Corti, however, is always limited to the basal turn. . . . The extent of the atrophic area of the organ of Corti is not related to age . . . there is no evidence that a damaged organ of Corti regenerates, so it must be due to chance alone that so many with this type of lesion are among the younger patients. Further study of this point may lead to a suggestion of the etiology. . . . We

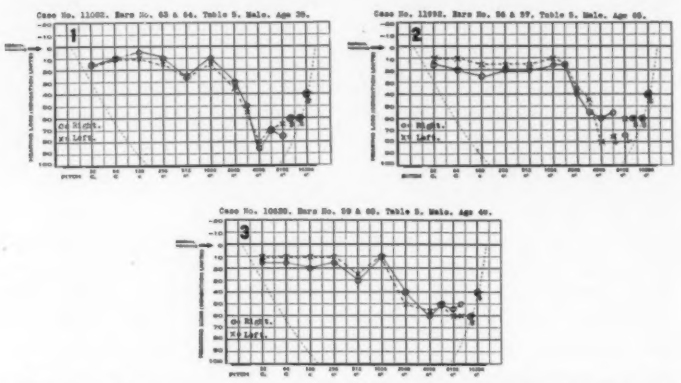

Fig. 11. Audiograms of three cases from the study by Crowe, Guild and Polvogt[40] entitled, "Observations on the Pathology of High Tone Deafness." These tests were made on the wards of the Johns Hopkins Hospital with a W.E. 1A audiometer. These writers classify these as "abrupt" high tone loss cases. No. 2 is listed as a boilermaker. The occupations of the others are not known.

have found extensive atrophy of the organ of Corti in but one female. The lesion is certainly more common in males. . . . Whether the total atrophy of the organ of Corti be extensive or localized, the external sulcus cells are always atrophic in the same area."

The description of the pathology given in this extensive study does not appear to differ materially from that of the much more limited studies by Habermann and Bruhl.

Two of the cases had histories of being employed in noisy surroundings; one was a boilermaker and the other a riveter. (The latter was tested with a 2A audiometer, and for this

reason is not illustrated here.) The histories, according to a personal communication by Dr. Guild, contain no note other than this concerning the occupations of their patients. The study was made to determine the correlation between structure and function and no etiological interpretation was attempted. There is no basis in their study for stating that the patients who showed an abrupt loss for high tones were deafened from occupational noises. Nevertheless, the similarity between their audiograms and many of those of the present study is very striking. Two other facts in their study apply here; first, the ages of the patients showing an "abrupt" drop is less than those showing "gradual" drop. This eliminates the possibility that this abrupt loss is an accompaniment of advancing age; second, only one ear from a woman patient was included in their study. In the present study, the records of women were in cases where there was a history of deafness from the use of the telephone or from blows on the ear. There is nothing in their study which can be interpreted as a denial that the "abrupt" tone loss results from acoustic trauma.

SUMMARY AND CONCLUSIONS.

This limited review of the literature was undertaken to determine the character of the deafness which, according to various investigators, is produced by occupational influences. The opinion of the majority of these writers, if correctly interpreted, gives the following clinical picture:

1. Bone-conduction is diminished.

2. The Rinné test gives the positive response.

3. In the Weber test, the tuning fork is lateralized to the less involved side.

4. There is a greater loss of acuity for high pitched tones than for low.

5. Paracusis Willisii is not a frequent accompaniment.

6. Tinnitus is not always present.

7. The tympanic membranes are usually intact.

8. The deafness is most marked immediately following the excessive stimulation. This is followed by a variable period

of recovery. The nature of this recovery is not fully described. Eventually the hearing loss may be so slight and so limited in extent that it may be unnoticed by the patient or discovered in routine hearing tests.

9. The insidious nature of the onset of the permanent impairment of hearing produced by excessive noise is such that those who are employed in these conditions are often unaware of any hearing defect.

10. That men are more frequently affected than women is the outgrowth of our social and industrial system.

In the absence of a history of possible occupational influences, these diagnostic points might equally well apply to almost any case of perceptive type deafness, regardless of its etiology. Thus it appears that occupational deafness has been inadequately described and it is justifiable to present additional evidence for differential diagnosis.

This present study is in agreement with the above described clinical picture. In addition, audiometric tests have revealed consistently certain characteristics in tests of cases of occupational deafness which appear to be diagnostic. These may be described as follows:

1. If a loss of auditory acuity has resulted from the effects of excessive stimulation of industrial noises, it is first evidenced by an abrupt dip or gap in the hearing range as determined by the audiometer, usually near c^5 (4,096 d.v.). Acoustic trauma should be suspected when these gaps appear. They are very commonly found in the records of the hearing of sportsmen and hunters who often have no loss in acuity for the spoken voice and are unaware of any hearing defect whatsoever.

2. With continued excessive stimulation, this gap in the curve becomes deeper and broader to include the tones above and below c^5. Eventually, the extremely high tones of the audiometer become inaudible and the curve shows an abrupt drop, usually between c^4 (2,048 d.v.) and c^5. Many times there is a tendency for the curves to approach the normal lines at c^6 (8,192 d.v.), with the deepest point in the gap near c^5. Artillerists, sailors who man the naval guns, and others who have been subjected to repeated excessive explosive sounds present

this type of record. If the loss does not extend downward to include c^4, these persons may note very little loss for the spoken voice. If the gap should extend downward to include c^4 and c^3, deafness for the spoken voice may be very pronounced.

3. Low tones (from c^2[512 d.v.] to the lower limit of audibility) are least often involved. Boilermakers and machinists who have been in these occupations for a long time sometimes show a loss in acuity for low tones, but the low tone loss is never as great as that for high tones.

4. The greatest loss consistently appears near c^5. Often there will be little or no loss for c^4. If hearing tests are made with tuning forks rather than with audiometers and the c^5 fork is not included in the set, many of these losses will not be discovered.

5. If the trauma to the ear results in the rupture of the tympanic membrane with subsequent suppurative otitis media, the contour of the hearing curves will be entirely different from those in which the deafness results from occupational noises alone. The same is true of cases of deafness in caisson workers where the deafness presumably is caused by the formation of gas bubbles within the cochlea.

Some pertinent facts which appear to be axiomatic may also be noted:

1. As with any hazard to life or health, all ears may not be equally affected by the same traumatic noise. Some may for a time escape without permanent injury. The greater loss of hearing in the left ear of hunters, sportsmen and infantrymen who shoot right-handed illustrates this point.

2. Explosive sounds and other loud noises within an enclosed chamber have a greater traumatic effect than similar noises in the open. When blasts are fired in a tunnel or caisson, the ears of the workmen need additional protection. Shooting galleries should be in the open. The locomotive engineer should not blow the whistle in a tunnel, train shed, or near a station where people are standing. The telephone receiver, with its very small air chamber, should be adequately protected from the effects of lightning and other high voltages.

3. Other occupational hazards to hearing than that of excessively loud noises, but which are truly occupational, exist in many industries. The locomotive engineer who at one moment leans far out of his cab and the next is subjected to the intense heat of the firebox may easily be subjected to repeated upper respiratory infections which may ultimately result in involvement of the middle ear. The laborer who works in the cold and damp of the tunnel or caisson may be similarly effected.

4. The medico-legal and compensatory implications are quite evident. This problem has not been approached in a scientific manner, and no just or adequate system of compensation has been established in this country. The fate of the deafened workman rests in the hands of a skilled lawyer, rather than in a just and sympathetic board or jury. In the same fashion, the employer may often be victimized by the unscrupulous pretender and his adviser. Justice can be attained through a program of repeated tests and a scientific study of the disability.

BIBLIOGRAPHY.

1. ANDREWS, A. H.: The Relation of Impairment of Hearing to Enginemen. *Railway Surg. Jour.*, XV, p. 58, 1908-9.

2. BACON, G.: A Manual of Otology. Lea Brothers and Co., Philadelphia, p. 418, 1902.

3. BALLA, A.: Impaired Hearing of Aviators: Professional Character. *Valsalva*, V, p. 397, 1929.

4. BALLENGER, W. L.: Diseases of the Nose, Throat and Ear. Lea and Febiger, Philadelphia and New York, Fourth Edition, p. 1010, 1914.

5. BARR, THOS.: Enquiry Into the Effects of Loud Sounds Upon the Hearing of Boilermakers and Others Who Work Amid Noisy Surroundings. *Proc. Glasgow Philosophical Soc.*, XVII, p. 223, 1886.

6. BARR, THOS.: Injurious Effects of Loud Sounds Upon the Hearing. *Brit. Med. Jour.*, p. 675, Sept. 20, 1890.

7. BASSOE, P.: Late Manifestations of Compressed-Air Disease. *Trans. 15th Int. Cong. of Hygiene and Demography.* III, Part 2, p. 626, 1912. Government Printing Office, 1913.

8. BAUER, L. H.: Aviation Medicine. Williams and Wilkins Co., Baltimore, p. 157, 1926.

9. BECK, K.: Noisy Occupations and the Ear. *Ztschr. f. Hals.- Nasen.- u. Ohren.*, XVIII, p. 387, 1927. *Abs. in. Arch. of Otolaryngol.*, VIII, p. 346, 1928.

10. BERNHARDT: Quoted in Kober and Hanson. Diseases of Occupation and Vocational Hygiene, p. 709.

11. BEZOLD and SIEBENMANN: *Textbook of Otology*, p. 272. Trans. by J. Holinger. E. H. Colegrove Co., Chicago, 1908.

12. *Op. Cit.*, p. 280.

13. *Op. Cit.*, p. 294.

14. *Op. Cit.*, p. 295.

15. BEZOLD, F.: Investigations Concerning the Average Hearing Power of the Aged. *Arch. of Otol.*, XXIII, p. 214, 1894.

16. BISHOP, S. S.: Diseases of the Nose, Throat and Ear. F. A. Davis Co., Philadelphia, Fourth Edition, p. 514, 1908.

17. BLAKE, C. J.: Influence of the Use of the Telephone Upon Hearing Power. *Arch. of Otol.*, XVII, p. 240, 1888.

18. BLAKE, C. J.: Occupational Injuries and Diseases of the Ear. See Kober and Hanson. Diseases of Occupation and Vocational Hygiene, p. 339.

19. BLEGVAD, V. RH.: The Influence of Their Calling Upon Telephone Operators, Particularly With Regard to the Hearing. *Abs. in Jour. of Laryngol. and Otol.*, XXIV, p. 172, 1909.

20. BOOT, G. W.: Caisson Workers' Deafness. *Ann. of Otol., Rhinol. and Laryngol.*, XXII, p. 1121, 1913.

21. BRAISLIN, W. C.: Two Cases of Injury to the Ear by Lightning. *Brooklyn Med. Jour.*, XX, p. 133, 1906.

22. BRAUNSTEIN: On the Influence of the Use of the Telephone on the Ear. *Abs. in Arch. of Otol.*, XXXIII, p. 531, 1904.

23. BRUHL, G.: Atlas and Epitome of Otology. W. B. Saunders and Co., Philadelphia, p. 114, 1902.

24. BRUHL, G.: Contribution to the Pathology of the Ear. Report of the 15th Meeting of the German Otological Society, 1906. See Reports of Societies, *Arch. of Otol.*, XXXVI, p. 91, 1907.

25. BRUNER, G.: An Interesting Case of Protracted Deafness Caused by the Report of a Musket. *Arch. of Otol.*, IX, p. 333, 1880.

26. BRYANT, W. S.: The Cochlea and Its Generalized Impressionability. *Trans. Amer. Laryngol., Rhinol. and Otol. Soc.*, p. 383, 1908.

27. BRYANT, W. S.: Prevalence of Ear Injuries and Diseases in the French Army. *Jour. of Laryngol. and Otol.*, XXXII, p. 338, 1917.

28. BUNCH, C. C., and RAIFORD, T. S.: Race and Sex Variations in Auditory Acuity. *Arch. of Otolaryngol.*, XIII, p. 423, 1931.

29. BUNCH, C. C.: Further Observations on Age Variations in Auditory Acuity. *Arch. of Otolaryngol.*, XIII, p. 170, 1931.

30. BUNCH, C. C.: Age Variations in Auditory Acuity. *Arch. of Otolaryngol.*, IX, p. 625, 1929.

31. CAPART: Diseases and Accidents of Telephone Operators. *Arch. internat. de Laryngol., d'Otol. et Rhinol.*, p. 209, May, 1911. *Abs. in* THE LARYNGOSCOPE, XXII, p. 656, 1912.

32. CARNEVALE-RICCI, R., and BASSI, U.: Professional Alterations of the Ear: Research With the Audiometer. *Medicine del lavoro.*, XXVI, p. 12, 1935.

33. CASTEX: Effects of Dynamite on the Ear. *Trans. Fr. Soc. of Otol. and Laryngol.*, 1893. *Abs. in Jour. of Laryngol. and Otol.*, VII, p. 307, 1893.

34. Ciocco, A.: Observations on the Hearing of 1980 Individuals: A Biometric Study. The Laryngoscope, XLII, p. 837, 1932.

35. Coggin, D.: A Curious Case of Deafness. *Boston Med. and Surg. Jour.*, CXVI, No. 1, p. 9, 1887.

36. Colledge, L.: Injuries to the Ear in Modern Warfare. *Jour. of Laryngol. and Otol.*, XXXVI, p. 283, 1921.

37. Connal, J. G.: Rupture of the Membrani Tympani with a Clonic Spasm of the Facial Muscles in a Diver. *Brit. Med. Jour.*, p. 719, Sept. 10, 1898.

38. Cooseman, E.: The Hearing Power of Beetlers. *Arch. of Otol.*, XXVIII, p. 413, 1899.

39. Cott, G.: Telephone Deafness. *Buffalo Med. Jour.*, LXXIII, p. 317, 1917-18.

40. Crowe, S. J.; Guild, S. R., and Polvogt, L. M.: Observations on the Pathology of High Tone Deafness. *Bull. of the Johns Hopkins Hosp.*, LIV, p. 315, 1934.

41. Culler, E., and Finch, G.: Effect Upon Cochlear Function of Intense Tonal Stimulation. *Amer. Jour. of Physiol.*, CXIII, p. 32, 1935.

42. Dalby, W. B.: Lectures: Diseases and Injuries of the Ear. *Lancet*, II, p. 873, 1872.

43. Davis, H., et al: Experimental Stimulation Deafness. *Science*, LXXXI, No. 20, p. 101, 1935.

44. Dench, E. B.: Diseases of the Ear. D. Appleton and Co., New York and London, Fifth Edition, 1919.

45. Dench, E. B.: Report of the New York Otological Society. Nov. 22, 1898. *Arch. of Otol.*, XXVII, p. 83, 1899.

46. Dickie, J. K. M.: Experiences of an Otologist in France. *Jour. of Laryngol. and Otol.*, XXXVI, p. 514, 1921.

47. Dore, G.: Deafness in Hunters. *Valsalva*, IX, p. 27, 1933.

48. Duane, A.: Middle- and Internal-Ear Disease Produced by the Concussion Due to Gun-Firing. *Arch. of Otol.*, XXIX, p. 62, 1900.

49. Egeblad, K.: Electrical Injury to the Organ of Hearing. *Ugeskr. f. Laeger*, II, p. 645, 1930. *Abs. in Psychol.*, Abs. VI, No. 10, p. 474, 1932.

50. Emerson, J. B.: Report of the New York Otological Society, Nov. 22, 1898. *Arch. of Otol.*, XXVIII, p. 83, 1899.

51. Erdman, S.: The Acute Effects of Caisson Disease. *Trans. of 15th Int. Cong. of Hygiene and Demography*, III, Part 2, p. 619, 1912. Government Printing Office, 1913.

52. Faulder, T. J.: Injuries to the Ear in Modern Warfare. *Jour. of Laryngol. and Otol.*, XXXVI, p. 277, 1921.

53. Field, G. P.: Diseases of the Ear. Lea Brothers and Co., Philadelphia, Fourth Edition, p. 296, 1893.

54. Fosbroke, J.: Pathology and Treatment of Deafness. *Lancet*, I, p. 645, 1830-31.

55. Fowler, E. P.: Marked Deafened Areas in Normal Ears. *Trans. Amer. Otol. Soc.*, XVIII, p. 262, 1928.

56. Fowler, E. P.: Limited Lesions of the Basilar Membrane. *Trans. Amer. Otol. Soc.*, XIX, p. 182, 1929.

57. FROMM, B.; NYLEN, O., and ZOTTERMANN, Y.: Electric Stimulation of the Cochlea. *Jour. Physiol.*, LXXX, p. 3P, 1933-34.

58. GELLE: Action of the Telephone on the Ear. *Soc. de Biol.*, June, 1, 1889. *Abs. in Arch. of Otol.*, XIX, p. 58, 1890. See also *Effets misibles de l'audition par le téléphon. Ann. des Mal. de L'orielle et du Larynx.*, XV, p. 380, 1889.

59. GILBERT, D. J.: Influence of Industrial Noises. *Jour. Ind. Hygiene*, III, p. 264, 1921-22.

60. GIRON: Occupational Deafness in Vineyard Workers. *L'Oto., Rhino., Laryngologic International*, April, 1922. *Abs. in Jour. of Laryngol. and Otol.*, XXXVIII, p. 95, 1923.

61. GOT, A.: War Deafness with Lesions of the Internal Ear. *Jour. of Laryngol. and Otol.*, XXXI, p. 374, 1916.

62. GRANT, D.: Varieties of Nerve Deafness. *Jour. of Laryngol. and Otol.*, XVII, p. 169, 1902.

63. GRAY, A. A.: *Jour. of Laryngol. and Otol.*, XXXVI, p. 384, 1921.

64. GREENE, J. O.: Cases of Injury to the Ear from External Violence. *Trans. Amer. Otol. Soc.*, V, p. 88, 1872.

65. GRIMWADE, S. W.: Occupational Diseases of the Nose, Throat and Ear. *Brit. Med. Jour.*, II, p. 890, 1925.

66. GROVE, W. E.: Otologic Observations in Trauma of the Head. *Arch. of Otolaryngol.*, VIII, p. 249, 1928.

67. GUILD, S. R.: Tests Upon Devices for the Prevention of War Deafness. *Anat. Rec.*, XIV, p. 36, 1918.

68. GUILD, S. R.: Otopathies of War; with Especial Reference to the Traumas Due to Air Waves. *Int. Assn. of Med. Mus. Bull.*, No. 7, p. 225, 1918.

69. HABERMANN, I.: Hardness of Hearing in Boilermakers. *A. f. O.*, Vol. XXX, p. 1. *Abs. in Arch. of Otol.*, XX, p. 171, 1891.

70. HABERMANN, I.: Occupational Deafness: A Continuation of the Investigations, etc. *Abs. in Arch. of Otol.*, XXXVI, p. 98, 1907.

71. HARRIS, T. J.: Diseases of the Internal Ear. Analysis of 351 Cases As Regards Prognosis and Treatment. THE LARYNGOSCOPE, II, p. 283, 1897.

72. HARTMANN, A.: Diseases of the Ear. Trans. by John Erskine. G. P. Putnam's Sons, New York, p. 32, 1887.

73. HEIMAN, TH.: A Blow on the Ear Followed by Death in a Week. *Arch. of Otol.*, XX, p. 17, 1891.

74. HILL, L.: Caisson Sickness. Longmans, Green and Co., New York, p. 77, 1912.

75. HOFAR, I., and MANTHNER, O.: Verletzungen des Ohres bei Katastrophalen Explosionen. Joseph Sofar. Wien., 1913. Reviewed by A. Tweedie in *Jour. of Laryngol. and Otol.*, XXVIII, p. 618, 1913.

76. HOLT, E. E.: Boilermakers' Deafness and Hearing in a Noise. *Trans. Amer. Otol. Soc.*, III, p. 34, 1882.

77. HORNE, J.: Gun Deafness and Its Prevention. *Lancet*, II, p. 462, 1914.

78. HORTON, G. P.: The Effect of Prolonged Acoustic Stimulation on the Auditory Sensitivity of Guinea Pigs. *Jour. Comp. Psych.*, XVIII, p. 405, 1934.

79. INGERSOLL, J. M.: The Otological Work in the U. S. A. General Hospital for Head Surgery. *Trans. Amer. Otol. Soc.*, XV, p. 68, 1919.

80. JACK, F. L.: *Arch. of Otolaryngol.*, VIII, p. 473, 1928.

81. JACKSON, C., and COATES, G. M.: The Nose, Throat and Ear and Their Diseases. W. B. Saunders Co., Philadelphia, 1929.

82. JOBSON, T. B.: Normal Gun Deafness. *Lancet*, II, p. 566, 1917.

83. Telephone Noises and Auditory Disturbances. *Jour. A. M. A.*, CIII, p. 282, July 28, 1934.

84. Effects of Noises on Organs of Corti. *Jour. A. M. A.*, CVIII, p. 415, Jan. 30, 1937.

85. JURGENS, E.: Affections of the Ear, Nose and Throat Following the Explosion of Bombs. *La Presse Otolaryngol.*, Part 5, 1907. *Abs. in Arch. of Otol.*, XXXVII, p. 305, 1908.

86. KEMP, E. H.: A Critical Review of Experiments on the Problem of Stimulation Deafness. *Psychol. Bull.*, XXXII, p. 325, 1935.

87. KERRISON, P. D.: Diseases of the Ear. J. B. Lippincott Co., Philadelphia and London, Fourth Edition, p. 131, 1930.

88. KIPP, C. J.: Report of the New York Otological Society, Nov. 22, 1898. *Arch. of Otol.*, XXVIII, p. 83, 1899.

89. KOBER, G. M., and HANSON, W. C.: Diseases of Occupation and Vocational Hygiene. Blakiston's Sons and Co., Philadelphia, p. 709, 1916.

90. KOBER, G. M., and HANSON, W. C.: Compressed-Air Illness, by S. Erdman, Chapter III, p. 187.

91. KOELSCH: Quoted by Kober and Hanson, p. 710.

92. KOETTER, A. T.: Report of a Case of Rupture of the Membrana Tympani While Using a Telephone. *Medical Fortnightly*, St. Louis, XXXVII, p. 99, 1910.

93. LAKE, R.: A Note on Boilermakers' Deafness. *Jour. Laryngol. and Otol.*, XXXVIII, p. 323, 1923.

94. LAKE, R., and PETERS, E. A.: Diseases of the Ear. William Wood and Co., New York, Fifth Edition, p. 265, 1927.

95. *Lancet*, II, p. 142, Aug. 4, 1866.

96. *Lancet*, p. 644, Oct. 2, 1886.

97. LEWIS, ROBT.: Deafness Due to Concussion from Gun Firing. *Arch. of Otol.*, XXIX, p. 63, 1900.

98. LICHTENBERG, K.: On Disturbances of Hearing in Railway Servants, With Reference to the Traveling Public. *Abs. in Jour. of Laryngol. and Otol.*, VI, p. 121, 1892.

99. LOEB, H. W.: Military Surgery of the Ear, Nose and Throat. *Med. War Manual*, No. 8, p. 31. Lea and Febiger, Philadelphia and New York, 1918.

100. LOVE, K.: Hearing in the Army. *Jour. Roy. Army. Med. Corps*, XXVII, p. 649, 1916.

101. MITCHIE, H. C.: Deafness; A New Method for the Determination of Defective Hearing. *Mil. Surg.*, LV, p. 49, 1924.

102. MOOS, S.: On the Diseases of the Ear in Locomotive Engineers and Firemen, Which May Endanger the Traveling Public. *Arch. of Otol.*, IX, p. 319, 1880.

103. Moos, S., and Steinbrugge, H.: Nerve Atrophy in the First Cochlear Convolution: Its Physiological and Pathological Importance. *Arch. of Otol.*, X, p. 1, 1881.

104. Mueller, R.: Effect of Artillery Practice on the Ears. *Arch. of Otol.*, XXVIII, p. 264, 1899.

105. McAuliffe, G. B.: The Essentials of Otology. Oxford University Press, New York, 1927.

106. McKelvie, W. B.: Weavers' Deafness. *Jour. Laryngol. and Otol.*, XLVIII, p. 607, 1933.

107. McMaster, G. T.: Gun Deafness: A Danger. *Amer. Med.*, IV, No. 8, p. 417, N.S., 1909.

108. Noel: Effect of Detonations Upon the Ear. *Abs. in Arch. of Otol.*, XX, p. 178, 1891.

109. Nimier: Effects Produced Upon the Ear by the Report of Firearms. *Abs. in Arch. of Otol.*, XIX, p. 58, 1890.

110. Packard, F. R.: Traumatic Perforations of the Membrana Tympani. *Jour. A. M. A.*, XXXIV, p. 1160, May 12, 1900.

111. Pierce, F. M.: The Telephone and Diseases of the Ear. *Brit. Med. Jour.*, II, p. 162, 1879.

112. Phillips, W. C.: Diseases of the Ear, Nose and Throat. F. A. Davis Co., Philadelphia, p. 188, Sixth Edition, 1924.

113. Politzer, A.: Diseases of the Ear. Lea and Febiger, Philadelphia, p. 175, Sixth Edition, 1926.

114. Politzer, A.: *Op. Cit.*, p. 638.

115. Politzer, A.: *Op. Cit.*, p. 716.

116. Rankin, D.: Occupational Diseases of the Ear, Nose and Throat, and Their Prevention. *Brit. Med. Jour.*, II, p. 891, 1925.

117. Report of the Medical Department of the United States Army in the World War. *Surgery*, XI, Part 2, p. 735 ff. Government Printing Office.

118. Ridout, G. B.: Gunfire Deafness in the Navy. *U. S. Nav. Med. Bull.*, XXVIII, p. 736, 1930.

119. Richardson, C. W.: Ear Protectors. *Trans. Amer. Otol. Soc.*, XIV, p. 525, 1918.

120. Richards, H.: Two Noteworthy Cases of Traumatic Rupture of the Membrana Tympani. *Trans. Amer. Otol. Soc.*, IV, p. 477, 1889.

121. Ridout, G. B.: Gunfire Deafness in the Navy. *U. S. Nav. Med. Bull.*, XXVIII, p. 736, 1930.

122. Robinson, J. R.: Functional Deafness and Destruction of the Membrana Tympani, Caused by an Electric Shock While Using a Telephone. *Ann. of Ophthal. and Otol.*, II, p. 45, 1893.

123. Roger, T. R.: Noise Deafness: A Review, etc. *Jour. of Laryngol. and Otol.*, XXX, p. 91, 1915.

124. Roger, T. R.: The Pathological Effects of Excessive Sounds on the Cochlear Apparatus Considered in Relation to Theories of Sound Perception. *Jour. of Laryngol. and Otol.*, XXXVIII, p. 66, 1923.

125. Roosa, D. B. St. J.: Remarks Upon the Diagnosis of Chronic Lesions of the Labyrinth or Acoustic Nerve With Illustrative Cases. *Trans. of Amer. Otol. Soc.*, IV, p. 34, 1887.

126. Roosa, D. B. St. J.: Diseases of the Ear. William Wood and Co., New York, p. 503, 1873.

127. Rugani, L.: Aural Lesions Due to Lightning. *Arch. Ital. di Otol., Rhinol. e Laringol.*, p. 353, Sept., 1911. *Abs. in* The Laryngoscope, XXII, p. 660, 1912.

128. Rutherford, Wm.: Tone Sensations With Reference to the Function of the Cochlea. *Lancet*, No. 2, p. 389, 1898.

129. Sachs, R.: The Hearing Power of Railroad Employees. *A. f. O.*, July, 1905. *Abs. in* The Laryngoscope, XIX, p. 216, 1909.

130. Scott, V. T.: Airplane Deafness and Its Prevention. *Mil. Surg.*, LII, p. 300, 1923.

131. Seely, A. C.: Importance of Eye, Ear, Nose and Throat Examinations With Railway Service. *Cal. St. Jour. of Med.*, VIII, p. 45, 1910.

132. Sexton, S.: Boxing the Ears. *Med. Rec., N. Y.*, XXXI, p. 649, 1887.

133. Sexton, S.: Injury of the Ear Caused by the Blast of a Bursting Shell, etc. *N. Y. Med. Rec.*, p. 201, Feb. 19, 1887.

134. Shambaugh, G. E.: A Restudy of the Minute Anatomy of Structures in the Cochlea, etc. *Amer. Jour. of Anat.*, VII, p. 245, 1907.

135. Shambaugh, G. E., and Knudson, V. O.: Report of an Investigation of Ten Cases of Diplacusis. *Trans. Amer. Otol. Soc.*, XVI, p. 397, 1923.

136. Shambaugh, G. E.: Year Book of Eye, Ear, Nose and Throat. Year Book Publishers, Chicago, p. 290, 1935.

137. Shuter, R. E.: Notes on War Injuries of the Ear. *Med. Jour. of Australia*, Sept. 29, 1917. Reviewed by A. J. Brady in *Jour. of Laryngol. and Otol.*, XXXIII, p. 255, 1918.

138. Simons, M. H.: Otological Experiences During the Battle of Santiago on Board the Battleship Iowa. The Laryngoscope, VI, p. 277, 1899.

139. Spear, E. D.: Case of Deafness Following a Blow. *Boston Med. and Surg. Jour.*, CXVI, No. 3, p. 71, 1887.

140. Stein, S.: Functional Investigation of Locomotive Hearing and Signal Hearing. *Abs. in* The Laryngoscope, VIII, p. 379, 1900.

141. Stevens, S. S.; Davis, H., et al: The Localization of Pitch Perception on the Basilar Membrane. *Jour. of Gen. Psych.*, XIII, p. 297, 1935.

142. Swann, C. C.: Effects of Noise on Hearing. *Internat. Jour. Med. and Surg.*, XLVI, p. 314, 1933.

143. Syme, W. S.: Diseases of the Nose, Throat and Ear. William Wood and Co., New York, p. 356, 1927.

144. Toynbee, J.: Diseases of the Ear. Churchill, London, p. 348, 1860.

145. Trible, G. P.: Comparative Hearing Requirements in Foreign and American Navies. *Mil. Surg.*, XL, p. 33, 1917.

146. Troena, F.: Changes in Hearing in Air Pilots: Statistical Study. *Valsalva*, IX, p. 337, 1933.

147. Turner, A. L.: Diseases of the Nose, Throat and Ear. William Wood and Co., New York, p. 365, 1924.

148. TURNER, A. L.: *Op. Cit.*, p. 364.

149. VAIL, H. H.: Traumatic Ear Conditions in Workers Under Compressed Air. *Trans. Amer. Otol. Soc.*, p. 139, 1929.

150. VITALI, S.: Professional Disease of the Ear With Particular Reference to Hearing. *Abs. in La medicina del Lavoro*, XXV, p. 109, 1934.

151. WESTMACOTT, F. H.: Occupational Diseases of the Ear, Nose and Throat, and Their Prevention. *Brit. Med. Jour.*, II, p. 886, 1925.

152. WEVER, E. G., and BRAY, C. W.: Auditory Nerve Impulses. *Science*, N. S., LXXI, p. 215, 1930.

153. WHITE, J. A.: The Telephone and the Ear. *Trans. Amer. Otol. Soc.*, XVIII, p. 171, 1928.

154. WILSON, J. G.: Injury to the Ear From High Explosives. *Trans. Amer. Otol. Soc.*, XIV, p. 239, 1917.

155. WILSON, J. G.: Effect of High Explosives on the Ear. *Jour. A. M. A.*, LXXI, No. 8, p. 628, 1918.

156. WOLF, O.: New Investigation Upon the Methods of Examination and the Disturbances of Hearing. *Arch. of Otol.*, IV, p. 267, 1874.

157. WOODRUFF, H. W.: Report of a Case of Rupture of the Tympanic Membrane From Indirect Violence. THE LARYNGOSCOPE, IV, p. 181, 1898.

158. YEARSLEY, M.: An Air Raid Case. *Jour. of Laryngol. and Otol.*, XXXII, p. 273, 1917.

159. ZILLIACUSIS: Disturbances of Hearing in Railway Employees. *Abs. in Arch. of Otol.*, XXXV, p. 502, 1906.

160. ZWAARDEMAKER, H.: Acoustic Railway Signals and Acuteness of Hearing. *Arch. of Otol.*, XXV, p. 385, 1896.

Oscar Johnson Institute.

SYMPOSIUM.

THE NEURAL MECHANISM OF HEARING.

B.—"NERVE DEAFNESS" OF KNOWN PATHOLOGY OR ETIOLOGY.

DEAFNESS FROM DRUGS AND CHEMICAL POISONS.*

DR. H. MARSHALL TAYLOR, Jacksonville, Fla.

Clinical and experimental studies have shown that there are a few drugs and chemicals that have a selective action for the auditory apparatus and can cause deafness. These studies have also shown that the VIIIth nerve is more frequently injured by foreign substances circulating in the blood than any other of the nerves of special sense. Idiosyncrasy to drugs is of paramount importance in the consideration of this subject, for the use of some drugs has shown that many persons have marked idiosyncrasies for them.

Pathologic studies have demonstrated that drugs may affect the auditory apparatus in various ways. They may have a definite toxic action on the ganglion cells of the cochlea and the associated nerve fibres. They may cause changes in the endothelium of the smallest capillaries or a contraction of the blood vessels of the internal ear, with resulting ischemia, lack of nutrition and anoxemia, followed by a degeneration atrophy of the ganglion cells and the nerve fibres in the basal coil of the cochlea. They may also cause a diminution in the endolymphatic pressure, with a resulting collapse of the membrana Corti.

Drugs may likewise cause injury to the cortical centre of hearing, some having an affinity for the protoplasm, others for the nuclei of the cells. In the study of deafness from drugs, perhaps more consideration has been given in animal experimentation to determination of the effect drugs may have on the spiral ganglion cells and nerve fibres of the cochlea than to their effect on the cortical center of hearing.

---

*Read as part of the Symposium, "The Neural Mechanism of Hearing," at the Seventieth Annual Meeting of the American Otological Society, Long Beach, N. Y., May 28, 1937.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 17, 1937.

Recently I had spinal punctures made on patients who had been given 30 gr. of quinine for the treatment of malaria and was interested to find quinine in the cerebrospinal fluid. Autopsies on the newborn, whose deaths were attributed to quinine used in the induction of labor, have frequently shown quinine in concentration in the brain. Sodium salicylate and alcohol also readily pass through the choroid plexus into the cerebrospinal fluid. The presence in this fluid of such agents as quinine, the salicylates and alcohol is not without clinical interest, and its importance has yet to be evaluated. It would be of interest to determine whether these drugs which pass through the choroid plexus into the cerebrospinal fluid do not also pass through the stria vascularis, which secretes the endolymph, and come in direct contact with the hair cells of the organ of Corti.

QUININE.

Of all the drugs that cause deafness, quinine is the most important. This drug has a definite toxic action on the spiral ganglion cells and seems to have a special affinity for the external hair cells in the basal turn of the cochlea. It not only acts as a protoplasmic poison, but also as a vasoconstrictor. From the resulting ischemia, lack of nutrition and anoxemia there may follow a degeneration atrophy of the ganglion spiralis and Corti's organ. Wittmaack[1] concluded that quinine induces a hypotonia of the labyrinth, a diminution in the endolymphatic pressure with resulting collapse of the membrana Corti.

Quinine is frequently used therapeutically as an abortifacient, in the induction of labor and in diseases concurrent with pregnanacy. According to Nicloux,[2] the human placenta acts as a dialyzer of drugs present in the maternal blood, and a crystalloid substance, such as quinine, passing through the epithelium of the placental villi into the fetal circulation, may be found there in relatively the same strength as in the maternal circulation.

The use of twilight sleep furnishes evidence that the fetal tissues react to drugs in the same manner as the maternal tissues. Because of the respiratory depression of the newborn child, its use has been largely abandoned in the practice of obstetrics. Numerous cases of congenital morphinism

furnish further evidence that drugs pass from the mother to the child through the placenta. Pettey[3] found that the newborn baby of a mother who is addicted to morphine or opium is as much an addict as the mother, for the child's blood and tissues are as fully saturated with the narcotic as are hers. He stated that every child born of a mother using opium should be given an opiate for the first three days after birth and appropriately treated for narcotism thereafter.

Quinine was found in the urine of 16 of the 17 babies examined by Dilling and Gemmel[4] after it had been given the mothers to induce labor. They reported finding the drug in the urine of the fetus six to 12 hours after the last dose had been administered to the mother. They observed that a concentration of quinine which may be toxic to the fetal tissues may persist in the fetus many hours after the wave of secretion in the maternal urine has subsided, owing probably to the slow return of the drug to the maternal circulation or to inability of the fetal kidneys to excrete it in such a concentration.

Regnier[5] stated that quinine was found in the blood and all the organs examined, including the brain, of a human fetus of four and one-half months 40 hours after the mother had taken 1.5 gm. of quinine sulphate. He further stated that the concentration was comparatively high in the blood, and higher in the brain than in the liver. In a case which resulted in a fetal death from quinine given the mother, King[6] found appreciable quantities of the drug, both in the brain and in the urine of the fetus.

The literature is replete with reports of cases of deafmutism that have been attributed to poisons in the mother's blood in diseases concurrent with pregnancy, such as rheumatic fever, influenza and malaria. Yet, little thought has been given to the fact that massive doses of drugs which cause nerve deafness are frequently prescribed to expectant mothers. In recent experiments in which he gave quinine and the salicylates to pregnant animals, Covell[7] found similar, but more profound, lesions in the hair cells of the organ of Corti of the fetal guinea pig than those of the adult animal.

When administered in the early months of pregnancy, drugs that cause nerve deafness may jeopardize the ear of

the unborn child. According to Dr. Streeter,[8] of the Department of Embryology of the Carnegie Institution of Washington, the spiral ganglion in the human embryo is recognized between the sixth and seventh week, and by the ninth week the cochlear nerve is definitely laid down, having its main characteristics, but it does not have its final myelinization until the last month of fetal life.

The presence of deafness at birth does not necessarily indicate that the condition is due to some hereditary defect, for it may result from injury to the organ of hearing in the course of the child's intrauterine life. This type of deafness should, therefore, be classified as deafness acquired in utero, not as hereditary deafness. The prevention of deafness should, then, begin with the prenatal life of the child, and that would necessarily include the proper care of the expectant mother.

That the otologist and the obstetrician have in the past ignored the significance of prenatal medication in infant deafness may be due to the difficulty of early diagnosis. A child must be age 2½ to 3 years before a diagnosis of nerve deafness can be made, and by that time the prenatal history has generally been dismissed. If the otologist, in obtaining the history of each child showing a nerve deafness, will inquire carefully into the history of drugs administered to the mother during her term of pregnancy, the etiology of a number of perplexing and otherwise unexplained cases of nerve deafness may be solved.

Quinine should be prescribed with the utmost caution for those persons who have an hereditary tendency to deafness and to early atrophy of the auditory nerve. It would seem that such medication might easily cause an early onset of deafness and a more rapid degeneration of the terminal filaments of the VIIIth nerve. In my opinion, quinine is definitely contraindicated for the pregnant women with a family history of deafness. Wilson[9] stated: "It is undoubtedly true that many of us disregard the action of drugs on susceptible individuals and on the unborn child. The more I see of progressive deafness, including otosclerosis, the more I am impressed with the deleterious effect of poisons carried by the circulation. Heredity counts undoubtedly, but circulatory disturbances supply the match to the gunpowder."

SALICYLATES.

Many persons are highly sensitive to the action of the salicylates, and on taking them may develop a slow, weak pulse, a subnormal temperature, dyspnea, dimness of vision, hissing and roaring sounds in the ear, and deafness. For severe degenerative changes to be produced in the auditory apparatus from the salicylates there must be a definite idiosyncrasy for them, as it is well known that many persons may take massive doses of these drugs without experiencing any disturbance of hearing. Permanent deafness may occur as a result of the use of the salicylates, but not so frequently as from quinine. Deafness from their use usually improves after their discontinuance, but does not always disappear.

The chief pathologic change in the auditory apparatus in poisoning from the salicylates is found in the nerve cells of the spiral ganglion. Wittmaack[1] found changes in the form of the cells, changes in and, in some instances, complete disappearance of the Nissl bodies, and in the more sensitive cases changes in the nucleus.

The possibility of the effect on the cortical centre of hearing must also be borne in mind in the administration of these drugs. Loberg[10] reported that sodium salicylate given to normal persons in doses of 6 gm. daily for six days was regularly found in the cerebrospinal fluid. To determine whether this drug passes through the stria vascularis into the ductus endolymphaticus and bathes the hair cells of the organ of Corti presents an interesting field for research. The fact that the salicylates are found in the cerebrospinal fluid suggests such a phenomenon.

The continued use of these drugs, particularly in children in the treatment of rheumatic fever and the exanthemata, may cause an impairment of hearing. It seems logical to assume that the deafness ofttimes attributed to the toxemias of these diseases may sometimes be due to the indiscriminate and excessive use of these drugs that are known to cause nerve deafness.

The possibility of damage to the fetal ear following the prolonged administration of the salicylates to the expectant mother cannot be ignored. Porak[11] reported finding sodium

salicylate in the urine of infants in cases in which this drug had been given to the mothers 30 minutes before delivery. He stated that the concentration was greater several hours after birth than at birth and that it was found in the urine for as long as three days. The fact that drugs affect mother and child alike is sufficient evidence that drugs which cause nerve deafness should always be administered with caution to the expectant mother.

ALCOHOL.

Alcohol evidently has less affinity for the VIIIth nerve than other poisons, such as quinine and the salicylates. It also has less affinity for the auditory nerve than for the optic nerve, for it is not so frequently affected in alcoholic patients. In most of the cases of deafness from alcohol the onset is sudden, deafness not infrequently developing within a few hours. Both ears are as a rule involved; tinnitus often persists for long periods of time, and the occurrence of vertigo indicates that the vestibular portion is somewhat, though not as markedly, involved as the auditory portion of the VIIIth nerve. Prognosis is doubtful. Complete abstinence from alcohol is the most important factor in treatment.

Animal experimentation showed that alcohol seems to reserve its selective action for the nuclei of the hair cells of Corti's organ, particularly in the basal coil, with only slight similar alterations in the hair cells of the vestibular apparatus.

Alcohol is one of the drugs that readily passes into the cerebrospinal fluid, and in alcoholic intoxication a larger quantity is found in the brain than elsewhere in the body, the hearing centre thus being exposed to a relatively large percentage of the poison. As the stria vascularis, which secretes the endolymph, seems to be analogous to the choroid plexus, which secretes the cerebrospinal fluid, it would therefore be of interest to determine whether or not alcohol may find its way into the endolymph as it does into the cerebrospinal fluid. Bárány and Rothfeld[12] concluded that disturbances of equilibrium in alcoholic intoxication have their origin in the cerebellar cortex.

Wade[13] reported five cases of deafmutism that were attributed to chronic alcoholism in the mother. In his experiments

on women in labor, Nicloux[2] gave alcohol in milk an hour before delivery of the child and was able to demonstrate it in the blood of the umbilical cord in practically the same concentration as in the maternal blood. In reporting experiments with alcohol and sodium salicylate, this author stated that the quantity in the fetal blood is always proportionate to the quantity in the maternal blood. The danger to the fetal ear in cases of chronic alcoholism in the expectant mother cannot be ignored, particularly so because the cochlear nerve does not have its final myelinization until the last month of fetal life.

TOBACCO.

A number of reports of impairment of hearing caused by tobacco occur in the literature. The symptoms are those of nerve deafness, often with associated disturbances of equilibrium, and in the less severe cases hearing tests are often negative. Symptoms usually disappear if the use of tobacco is abandoned. Pharyngitis, so common in smokers of tobacco, and the constant irritation of the mucous membranes of the nasopharynx and the Eustachian tubes that occurs in heavy smokers, have in the past been suggested as causing symptoms of the middle ear without producing any actual toxic effect of the tobacco on the ear. More recently, cases with evidence of damage to the inner ear have been reported.

In his series of cases in which tobacco was used excessively, Delie[14] found deafness the chief symptom, and it was especially severe in young patients with otosclerosis. He felt that the excessive use of tobacco should be avoided by persons with any disease of the ear, particularly by young persons with beginning otosclerosis or hereditary tendency to deafness. He concluded that tobacco acts by stimulation of the sympathetic nerves, which results in an anemia in the auditory nerve and especially in the ganglia and nerve fibres of the cochlea, the altered circulation finally leading to faulty nutrition of the nerve, with resulting degenerative changes. Referring to Delie's theory of the mechanism of the action of tobacco on the VIIIth nerve, Gy[15] noted that the direct effect of tobacco on the blood vessels should not be overlooked; he concluded that tobacco causes a true trophoneurosis, which results in atrophic neuritis of the auditory nerve.

Otological findings in a series of cases of toxic amblyopia resulting from the excessive use of both alcohol and tobacco, reported by Carroll and Ireland,[16] pointed to islands of deafness comparable to the island defect in the visual field. As the average loss of hearing for the spoken voice in these cases was only 8 per cent, all were not aware of their deafness, but a considerable number of these patients showed a perceptible reduction of useful hearing. Since the ophthalmologic lesion is found in the retinal ganglion cells and the spiral ganglion cells and the ganglion cells of the retina are functionally and anatomically similar, these authors concluded the otologic lesion occurs in the spiral ganglion cells.

The animal experimentation of Schroeder and Hinsberg[17] showed changes in the cells of the spiral ganglion, due to tobacco, that were similar to changes in other nerve tissues, including the brain. Other experiments on animals showed injury to the cells of the vestibular ganglion. Kostencki,[18] recently reporting experiments on rabbits, concluded that nicotine acts on the entire vascular system of the hearing apparatus, including both the central and the peripheral portions, while alcohol exerts its toxic effect only on the central portion.

ARSENIC.

With the advent of salvarsan, arsenic has come into prominence as a drug that may affect unfavorably the auditory function. Deafness and tinnitus have often been reported after its continued use. Animal experimentation has shown definite changes in both the vestibular and cochlear branches of the VIIIth nerve following its administration. Arsenic is one of the drugs that not only has effect on the ganglion cells, but also Reissner's membrane, which may change the endolymphatic pressure. Miyamote[19] sectioned the internal ear of 13 guinea pigs after having given them toxic doses of atoxyl, an arsenic preparation. In six of these animals there was marked degeneration of the epithelial layer of the stria vascularis, with a sinking in of Reissner's membrane, resulting in narrowing or complete obliteration of the lumen of the cochlear duct. This finding the author attributed to failure of the normal secretion of the endolymph. In all but one of the animals in which the changes described in the stria vas-

cularis were present, the ganglion cells and Corti's organ also showed advanced changes.

The question of the effect of salvarsan on the auditory nerve is still open. Evidences of injury to this nerve have been noted in cases of syphilis under treatment with salvarsan, and the question arises whether the injury results from the effect of syphilis or from salvarsan. There is, however, some evidence that this drug, under conditions not well understood, may have a toxic action on the auditory nerve, as well as other nerves. A few cases of injury to this nerve have been reported when salvarsan was used in nonsyphilitic cases. Injury to it from neosalvarsan has not been reported.

LEAD.

Of the industrial poisons that may affect the ear, lead is one of the most important. It is known to have a toxic action, not only on the nerve parenchyma, but also on the blood vessels. Changes in the latter appear to play the chief role. There is some difference of opinion as to whether deafness resulting from lead poisoning is of central origin, due to vascular changes, or the result of direct effect on the cells of the spiral ganglion. The fact that, as a rule, there is a diminution in hearing for the high notes points to a destruction of the cells at the basal turn of the cochlea. It is doubtful whether either the otologist or the internist has given sufficient attention to lead poisoning as a possible cause of nerve deafness.

PHOSPHORUS.

Another one of the industrial poisons that produces deafness is phosphorus. Poisoning from this substance has long been observed to cause tinnitus, vertigo and deafness. This condition is frequently found in workmen exposed to its fumes in making friction matches. Experiments with rats and mice poisoned by phosphorus showed circulatory disturbances in the region of the temporal bone, but no necrosis of the bone itself. Beck[20] found evidence of changes in the cells of the spiral ganglion in rabbits poisoned by this element. Nissl,[20] in his studies of its effects on the cells of the brain, found primary changes in the protoplasm, rather than the nucleus of the cell, as was the case in the spiral ganglion cells in Beck's experiments with it.

CARBON MONOXIDE.

Vertigo, tinnitus and deafness have been noted as symptoms of acute carbon monoxide poisoning. This type of poisoning is usually the result of inhaling illuminating gas and is due to the carbon monoxide content of the gas. Deafness from this cause is usually temporary, but permanent impairment of hearing has not infrequently been reported. It seems to be the consensus that carbon monoxide may cause changes in both the cochlear and vestibular branches of the VIIIth nerve and also injure the vestibular nucleus.

Rutenberg,[21] reporting experiments on mice, guinea pigs and cats poisoned by inhalation of carbon monoxide, stated that a study of microscopic sections of the inner ear, stained with hematoxylineosin or Weigert's stain, showed some hemorrhage in the scala tympani and the inner border of the lamina basilaris membranacea, and slight hemorrhage in the scala vestibuli, but no other changes, either in the cochlea or in the vestibular apparatus. Ruttin[22] reported three cases of deafness from acute carbon monoxide poisoning. In one there was tinnitus and slight loss of hearing, with diminution of bone conduction, indicating a cochlear lesion. In another, deafness was much more marked, being total on the left side, with a negative Rinné's test and the interval much shortened in Schwabach's test. In the third case deafness was complete. In all, there were disturbances of the vestibular reactions; in one they were entirely abolished. He concluded that carbon monoxide gas acts on the cochlear and vestibular ganglia.

CARBON DISULPHIDE.

A temporary deafness, vertigo and tinnitus are not infrequent in carbon disulphide poisoning, and auditory hallucinations also occur. This type of poisoning is frequently seen among workmen in the rubber industry. The deafness is usually transitory and disappears after the subsidence of other toxic symptoms.

OIL OF CHENOPODIUM.

Oil of chenopodium is used as an anthelmintic, particularly in the treatment of the ascaris lumbricoides. It continues to

retain its popularity in some sections of this country. Deafness from this drug has frequently been reported. It is associated with other toxic effects of the drug, such as tingling in the feet and hands, severe headache, dizziness, vomiting, nausea and occasionally convulsions. It varies from slight impairment of hearing to a complete bilateral deafness, which, in some cases, becomes permanent. The onset of the deafness is rapid, occurring within two hours in severe cases.

It has been claimed that nearly one-third of the patients given oil of chenopodium in therapeutic doses show some disturbances of hearing. A comprehensive review of the American and European literature revealed no record of animal experimentation to determine what part of the auditory apparatus the oil of chenopodium affects. There is ample evidence, however, in clinical cases to show that the deafness is of the type affecting the internal ear. Suzuki[23] cited several Japanese authors whose experiments showed that this drug damages the spiral ganglion cells primarily and, secondarily, the nerve fibres of Corti's organ.

MERCURY.

Mercurial poisoning occasionally causes disturbances of the inner ear and Ménière's symptom complex. Wyss[24] reported the cases of two chemists who suffered from chronic mercurial poisoning and were both deaf and blind before death. Several observers have reported symptoms of labyrinthine involvement and deafness associated with this poisoning. When mercury is used in the treatment of syphilis the question arises, as with salvarsan, whether or not any involvement of the auditory nerve is due to syphilis or the drug.

MORPHINE.

Infrequently tinnitus and deafness occur after the subcutaneous administration of morphine. This drug is recognized as among the drugs which may, though rarely, have a deleterious effect on the auditory apparatus. This effect is manifested by auditory hallucinations, tinnitus and loss of hearing. Politzer[25] listed morphine among the drugs that may cause deafness.

ANILIN DYES.

Numerous authors have reported deafness from poisoning by anilin and anilin dyes. It may be accompanied by tinnitus, often annoying and persistent, and vertigo, which are to be regarded as of central origin in the opinion of Beck.[20] This condition is attributed to the toxic action of the methemoglobin formed by anilin. Other poisons that form methemoglobin also may cause pathologic changes in the VIIIth nerve. Deafness from this cause is characterized by a decrease both of air and bone conduction, with a positive Rinné.

Of particular interest is poisoning by paraphenylendiamine, a derivative of anilin used in hair dyes, which produces labyrinthine symptoms. In two of the 18 cases reported by Laurens,[26] the onset of symptoms was rapid, vertigo, marked tinnitus and some diminution of hearing by air conduction following soon after application of the dye. In the others it was more gradual, occurring after prolonged use. Tinnitus, sometimes accompanied by attacks of vertigo, especially after fresh application of the dye, and progressive loss of hearing resulted. The deafness was characterized by diminution of both air and bone conduction with a positive Rinné, the symptoms in many respects resembling those of otosclerosis. Some of these cases presented general symptoms indicative of poisoning, such as neuralgic pains, headache and weakness.

A fur dyed with paraphenylenediamine and worn almost daily was the cause of a case of poisoning by anilin reported by Watson-Williams.[27] Tinnitus, at first occasional, became incessant and was accompanied by vertigo; severe nerve deafness also developed. In about 10 weeks after the fur was discarded the symptoms disappeared.

SUMMARY.

Certain drugs and chemicals have a selective action for the auditory apparatus and may also injure the cortical centre of hearing.

Idiosyncrasy for drugs may be an important causative factor of nerve deafness.

Owing to the permeability of the placenta to drugs causing nerve deafness, their administration during pregnancy may jeopardize the ear of the unborn child.

Chief among these drugs is quinine, used frequently as an abortifacient, in the induction of labor and in the treatment of diseases concurrent with pregnancy.

The salicylates and alcohol also frequently cause deafness; and, less frequently, tobacco, arsenic, lead, phosphorus, carbon monoxide, carbon disulphide, oil of chenopodium, mercury, morphine and anilin dyes.

Drugs and industrial poisons that are capable of damaging the internal ear merit greater consideration and deserve more careful study as etiologic agents of deafness.

REFERENCES.

1. Wittmaack, K.: Ueber Chinin wirkung im Gehoerogan. *Zeitschrift. f. Hals-, Nasen- u. Ohrenheilk.*, 39:211-222, 1936.

2. Nicloux, M.: Le passage des substances chimiques de la mere au foetus. *Obstet.*, 2:840, 1909.

3. Pettey, G. E.: Congenital Morphinism, with Report of Cases. *South. Med. Jour.*, 5:25, Feb., 1912.

4. Dilling, W. J., and Gemmell, A. A.: A Preliminary Investigation of Foetal Deaths Following Quinine Induction. *Jour. Obstet. and Gynaec.*, Brit. Emp., 36:353, 1929; Further Investigations of the Death of the Child Following Induction of Quinine. *Ibid.*, 37:528, 1930.

5. Regnier, M. T.: De la permeabilite placentaire aux substances medicamenteuses et toxiques: quinine. *Compt. rend. Soc. de biol.*, 120:1089-1090, 1935.

6. King, E. L.: Does Quinine in the Induction of Labor Have a Deleterious Effect on the Fetus? *Jour. A. M. A.*, 101:1145, Oct. 1, 1933.

7. Covell, W. P.: A Cytologic Study of the Effects of Drugs on the Cochlea. *Arch. Otolaryngol.*, 23:633-641, 1936.

8. Streeter, G. L.: Personal communication to the author.

9. Wilson, Gordon: Personal communication to the author.

10. Loberg, K.: Eine klinische Methode zur quantitativen Bestimmung von Salizylsaeure im Blutserum und Liquor cerebrospinalis. *Biochem. Zeitschrift.*, 170:173, 1926.

11. Porak, C.: L'absorption des medicaments par le placenta. Paris: G. Masson, 1878. *Jour. de Therap.*, 5:332 and 412, 1878.

12. Barany, R., and Rothfeld, J.: Untersuchungen des Vestibularapparates bei akuter Alkolintoxikation und bei Delirium tremens. *Deutsche. Zeitschrift. f. Nervenheilk.*, 50:133, 1913.

13. Wade, W.: *Amer. Ann. Deaf.*, 57:284, 1912.

14. Delie, M.: Horstörungen zur Tabakmissbrauch. *Internat. Centralbl. f. Ohrenheilk.*, 3:34, 1904-1905.

15. Gy, A.: La vision et l'audition dans l'intoxication tabagique chronique. *Rev. internat. d'hyg. et de therap. ocul.*, 7:49, 1913.

16. CARROLL, F. D., and IRELAND, P. E.: Association of Toxic Deafness with Toxic Amblyopia Due to Tobacco and Alcohol. *Arch. Otolaryngol.*, 21:459, 1935.

17. SCHROEDER, P., and HINSBERG, V.: Zur Frage der spezifischen Wirkung von Chinin und Salizylsäure aud des Ganglion spirale. *Zeitschrift. f. Ohrenheilk.*, 73:65, 1915-1916.

18. KOSTENCKI, A.: Experimentelle Untersuchungen über die Schädigung des Gehörapparates durch Nikotin. *Beitr. z. prakt. u. theoret. Hals-, Nasen- u. Ohrenheilk.* (Passow-Schäfer), 31:451, 1935.

19. MIYAMOTE, T.: Experimentelle Untersuchungen über dis Schädigung des Gehörorgans durch die Giftwirkung der Arzenimittel. *Arbeuten a. d. med. Universitat. z. Okayama*, 2:412, 1931.

20. BECK, K.: Experimentelle Untersuchungen über den Einfluss von Bakterientoxinen und Giften auf das Gehörorgan. *Zeitschrift. f. Ohrenheilk.*, 68:128, 1913; Intoxikationen. *Handb. der Hals-, Nasen- u. Ohrenheilk.*, Berlin: Springer, 6:828-841, 1926.

21. RUTENBERG, D. N.: Die veränderung des statischen und des Gehörapparates bei CO-Vergiftung. *Arch. f. Ohren-, Nasen- u. Kehlkopfh.*, 124:3, 1929.

22. RUTTIN, E.: Ueber Schädigung des Gehörorgans durch Gasvergiftung. *Zeitschrift. f. Ohrenheilk.*, 77:60, 1918; Zur Klinik der Schädigung des Gehörorgans durch Leuchtgasvergiftung. *Monatsschr. f. Ohrenheilk.*, 68:449, 1934.

23. SUZUKI, Y.: Experimentelle Studien über den Einfluss von Extractum Filicis und anderer Gift auf das Gehörogan. *Mitt. a. d. med. Akad. zu Kioto*, 17:984-988, 1936.

24. BENESI, O., and SOMMER, J.: Neuritis toxica nervi octavi. *Handb. der Neurolog. des Ohres.*, Berlin, 2:787-860, 1929.

2'. POLITZER, A.: A Textbook of Diseases of the Ear. Philadelphia: Lea & Febiger, 1926.

26. LAURENS, P.: Un poison du labyrinthe paraphenylenediamine. *Bull. d'oto-rhino-laryngol.*, 20:161, 1922.

27. WATSON-WILLIAMS, E.: Paraphenyl-diamine labyrinthitis. *Lancet*, 1:1123, 1928.

111 West Adams Street.

BOOK REVIEW.

The Larynx and Its Diseases. By Chevalier Jackson, M.D., Sc.D., LL.D., F.A.C.S., Professor of Bronchoscopy and Esophagoscopy, Temple University, Philadelphia; and Chevalier L. Jackson, A.B., M.D., M.Sc. (Med.), F.A.C.S., Professor of Clinical Bronchoscopy and Esophagoscopy, Temple University, Philadelphia. 55 pages with 221 illustrations, including 11 plates in color. Philadelphia and London: W. B. Saunders Co., 1937. Cloth, $8.00 net.

This volume on the larynx fills a very definite need for colored photographs of laryngeal pathology. The artistic capabilities of the senior Jackson enable him to put his clinical findings in oil and paint with the technique of Leonardo da Vinci.

The vagaries of abductor paralysis have been fully covered in a way that beggars description. The explanation of difficulty in breathing in this condition as a "cellar-door" inspiratory blast and the description of how these patients adjust themselves by less vigorous breathing, is indeed unique.

A splendid article on laryngo-tracheo-bronchitis, with colored photographs, will help considerably those of the profession who are inclined to call these cases diphtheria.

The dull legends usually accompanying cuts of microphotographs are missing; here we have clear cuts with descriptive legends that leave nothing to be desired for clarity and understanding.

Laryngeal symptoms seen in general medical practice, as well as many borderline cases, with excellent drawings and colored photographs, are ably presented.

The method of "coring" a strictured larynx and trachea will doubtless obviate many surgical procedures. This chapter alone, to the reviewer's mind, makes a distinct advance for peroral laryngoscopy.

The chapter on contact ulcer of the larynx is a very definite contribution of the Jacksons to Laryngology. Here again the subject is easily grasped and beautifully illustrated.

In their previous books tracheotomy has been well handled, but one should not neglect reading what they have to say about tracheotomy and intubation in the present volume.

The authors' statement that the contents of their book has been thoroughly tested in the crucible of their clinical experience is obvious, and every physician who is interested in laryngeal disease should use this volume as reference.

E. L. M.